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STRANGULATED HERNIA*

A Report of Two Cases in Which the Sac Was Found In An Unusual Location

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WHEN the sac of a strangulated hernia is in an unusual location the difficulties in establishing the diagnosis and the subsequent mortality are greatly increased. Such is the case when strangulation develops in an interparietal hernia. In this condition the sac dissects between the layers of the abdominal wall instead of becoming subcutaneous. This type of hernia is probably more frequent than is generally realized. Many cases are overlooked or are mistaken for abdominal or retroperitoneal tumors. The following case reports are illustrative of the difficulties which may be encountered.

CASE REPORTS

Case 1. A. B., a 57-year-old Jew, entered the Peter Bent Brigham Hospital complaining of abdominal pain, vomiting, distention and obstipation of 24 hours' duration. Six years previously the patient had had a course of medical treatment for duodenal ulcer with complete relief of symptoms. Four years before the present admission he had had an attack of severe, cramp-like abdominal pain. The pain was situated in the lower abdomen slightly to the left, and was subsequently associated with nausea, vomiting and abdominal distention. The patient entered another hospital where an x-ray examination of the gastrointestinal tract by means of a barium meal and enema was reported to be negative. A diagnosis of chronic constipation and possible intestinal obstruction of unknown etiology was made. Operation was not thought necessary and the patient was discharged improved. Since that time he had had recurrent bouts of pain, all quite similar in character but less severe than the initial attack. The attack which brought the patient to this hospital was more severe than usual. The pain was accompanied by nausea, vomiting and abdominal distention. Always constipated, the patient felt that this symptom had been more severe in recent months. The last bowel movement was just prior to the onset of the attack.

Physical examination on admission showed a well-developed, slightly obese man in moderate distress. The abdomen was moderately distended and tympanitic. In the left lower quadrant there was a tender mass about 10 cm. in length and 6 cm. in width. The external inguinal rings admitted the examining finger, but there was no tenderness, palpable mass or impulse on coughing. Digital examination of the rectum was negative. The temperature was 99°F. by rectum, the pulse 90 and the blood pressure 130/80. The white-blood-cell count was 20,600, with 83 per cent polymorphonuclear leukocytes. The red-blood-cell count was 5,000,000, and the examination of the urine was negative.

The diagnosis on admission was intestinal obstruction due to neoplasm of the sigmoid. However, the surgical consultant suggested a diagnosis of a left inguinal hernia which had dissected laterally and upward toward the flank between the internal and external oblique muscles. Further confirmation of this impression was obtained by an x-ray examination which showed evidence of dilated loops of small intestine in a plain film and an apparently normal large bowel by means of a barium enema.

At operation an oblique incision similar to but higher and longer than that usually used for inguinal herniorrhaphy was made. On opening the fibers of the external oblique muscle a tense bluish mass, somewhat irregular in shape, was exposed. This was easily delivered, and proved to be a large hernial sac which had dissected upward and laterally from the internal abdominal ring between the internal and external oblique muscles. The sac contained a strangulated loop of small bowel and omentum. The latter showed extensive necrosis with much old scarring and chronic fat necrosis, indicative of previous attacks of strangulation. As the bowel was viable it was returned to the abdomen. The sac and omental tissue were excised and the neck of the sac closed by high ligation. The inguinal canal was repaired without transplantation of the spermatic cord. The postoperative course was uneventful. There has been no recurrence of symptoms to date, 1 year later.

Case 2. C. M., a 40-year-old farmer, complained of a chronic pain in the right lower quadrant associated with the appearance of a mass in that area. Some years before, the patient had had an appendectomy with drainage but had never noticed any weakness or bulging in the wound, and the present palpable mass was lateral to the scar of the operation. When first seen, the patient was thought to have a retroperitoneal tumor. He was advised to enter the hospital for diagnostic procedures. This he refused to do. One month later he entered because of increasing pain and vomiting of 48 hours' duration.

Physical examination on admission showed an obviously

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ill man in severe pain. The abdomen was distended, tympanitic and tender. There was an irregular puckered scar of an incision in the right lower quadrant with the scar of a drainage site about 5 cm. lateral to it. There was a mass 7 cm. lateral to the scar of the drainage site, well away from the incision. It extended from the groin upward and outward toward the right flank. At first it seemed to be retroperitoneal, but the physical examination suggested that the lesion might be more superficial. The clinical picture was obviously that of intestinal obstruction, but the mass could be correlated with this diagnosis only on the assumption that it was a ventral hernia arising from the drainage site and dissecting laterally between the layers of the abdominal wall.

At operation this was found to be the case. Between the internal and external oblique muscles, lateral to the old incision, there was a large hernial sac under acute tension. The neck of the sac could be traced to the site of the old drainage wound. The sac was found to contain a loop of gangrenous small bowel and omentum. Resection of the non-viable segment of bowel with end-to-end anastomosis was performed. The convalescence, except for a slight infection in the wound, was not remarkable.

It is interesting that in both these cases the clinical picture was misleading. In one case it suggested a large-bowel lesion, and in the other a retroperitoneal tumor. In fact, these were the diagnoses of several observers. The correct diagnosis was arrived at largely by a proper interpretation of the physical examination, which in both cases suggested that the tumor was in the abdominal wall rather than retroperitoneal or intraperitoneal. A knowledge of the various types of interparietal hernia is a valuable aid in the correct appraisal of the physical examination.

Interparietal hernias are usually of the indirect inguinal type. However, direct or, as in one of the cases reported above, ventral hernias occasionally become interparietal. Interparietal hernias may be divided into three anatomical groups: properitoneal, interstitial and inguinosuperficial.[1] In the properitoneal type the sac lies between the peritoneum and the transversalis fascia; in the interstitial it lies between the muscles of the abdominal wall; and in the inguinosuperficial it lies between the external oblique aponeurosis and the skin. This latter type is the commonest and is usually quite easily recognized. In the interstitial and properitoneal types the diagnosis is more difficult, and it is in these cases that the hernia is likely to be mistaken for an intra-abdominal or retroperitoneal tumor.

Properitoneal and interstitial hernias are often composed of two sacs which are hourglass in shape. One may occupy the usual position in the inguinal canal and the other extend laterally between the layers of the abdominal wall. There is always a common opening into the abdomen at the neck of the sac. The size of the external inguinal ring in these cases may be normal or unusually small.

SUMMARY

Two cases of strangulated hernia in which the sac was found in an unusual location (interparietal) are reported. In one of these the sac arose from the internal abdominal ring and passed between the internal and external oblique muscles in a lateral and upward direction. In the other the sac arose at the site of an appendectomy drain and extended laterally into the flank between the same muscles. Familiarity with the various types of interparietal hernia is a valuable aid in the diagnosis and leads to early surgical intervention and a favorable outcome of what otherwise is a highly fatal condition.

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THE FEMALE SEX HORMONES*

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THE sex cycles of women have been known from time immemorial — the twenty-eight day, menstrual, frustrated or infertile cycle and the two-hundred-and-eighty-day, reproductive or fertile cycle.

Between 1900 and 1915 the anatomical basis of the cycles was studied intensively. Growth, maturation of the ovum, ovulation and corpus luteum formation were synchronized with the changes which take place in the uterine mucous membrane. It was determined that during the ovarian follicular phase the mucosa proliferates and during the corpus luteum phase it secretes.

In the succeeding decade, physiological experiments demonstrated that these changes were due to hormones elaborated by the follicle and corpus luteum. Shortly afterward, the primary stimulus of the ovary was discovered to be centered in the anterior pituitary gland. Since then, successful efforts have resulted in the production of crude gonadotropic extracts — that is, testicular and ovarian stimulating factors — as well as isolation of pure, crystalline follicular (estradiol) and corpus luteum (progesterone) hormones.

It should be mentioned that the sex hormones, both male androgenic (testosterone), female estrogenic (estradiol), progestational (progesterone) and adrenocortical (adrenosterone), are very similar in structure; that they or some of their derivatives can be changed into each other by chemical means and that they all are closely related to cholesterol. Their generic chemical name is "sterid" hormones. The importance of this relation is significantly emphasized by the fact that women as well as men excrete both male and female sex hormones. Disturbances in the balance of this normal chemical hermaphroditism may produce serious disturbances.

By means of biological reactions obtained in animals, and in some cases by chemical tests, the presence of these hormones in the blood, urine and tissues has been determined, both qualitatively and quantitatively. This has enabled investigators to demonstrate the underlying hormonal causes which produce the sex cycles (menstrual and reproductive).

In women no cyclical phenomena appear until the onset of puberty. The menstrual, abortive cycle is initiated by the gonadotropic secretion of the prepituitary, which reaches its acme in the blood stream between the ninth and eleventh days. This hormonal flow stimulates ovarian follicular growth. The growing follicle, in turn, as does the corpus luteum, secretes estradiol, which stimulates the uterine mucosa to proliferate. When ovulation occurs (eleventh to fourteenth day) the corpus luteum secretes estradiol and progesterone. The latter causes secretion of the uterine mucosa, a change essential for successful embedding of the fertilized ovum.

If impregnation is not achieved, the corpus luteum regresses, the secretion of hormones by the ovaries ceases, and bleeding (menstruation) takes place through the degenerating mucosa. If pregnancy should ensue, the corpus luteum persists, and with it a high level of gonadotropic, estrogenic and progestational hormones continues in the blood throughout pregnancy. As a result, the many growth changes in the genital sphere which we associate with pregnancy develop and are maintained.

Hormonal studies performed on hundreds of women have given a welcome and enlightening insight into the hormonal conditions, both in normal states and in functional disease.

Today, however, a complete hormonal study of even one patient has become a formidable task. It entails weekly assay of gonadotropic and estrogenic factors of the blood, and daily assay (in three-day batches) of gonadotropic, estrogenic, corpus luteum and androgenic factors of the urine. The norms for fertile, menstruating women show some variation within well-defined limits. Additional data are obtainable by means of suction biopsies from the uterus and by vaginal smears.

Our investigations have shown that the pituitary–follicular–corpus-luteum hormonal mechanism either functions normally or, if disturbed, overfunctions or underfunctions exactly as do other glands of internal secretion.

OVERFUNCTION

Overfunction, as evidenced by a greatly increased excretion of estrogens in the urine but no increase in the blood level, is observed in functional menorrhagia and metrorrhagia. Stationary hyperplasia of uterine mucosa and hyperplasia of the uterine muscle result.

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In contradiction to the numerous reported favorable results with gonadotropic factor (Follutein, Antuitrin S or anterior-pituitary-like hormone) I have seen no improvement from the use of these drugs. The results obtained with androgens, recently recommended, have likewise proved disappointing.

The treatment of menorrhagia and metrorrhagia must be varied according to the age group in which it occurs.

In adolescents from eleven to sixteen years old, and young women of sixteen to twenty years, if the basal metabolism is below −15 per cent (often −35 to −25 per cent), thyroid extract in adequate dosage is curative. If the thyroid function is normal, moccasin venom (1:3000) given subcutaneously in doses increasing from 0.2 to 2 cc. thrice weekly controls the bleeding.

In mature women, non-endocrine causes of bleeding, such as fibroids, threatened or incomplete abortion, adnexal inflammation, ectopic gestation and cervical and corpus carcinoma, must first be excluded. Ergot, Cotarnine Phthalate and Antuitrin S may be tried but rarely prove effective. Resort should then be had to curettage, the curettings being subjected to microscopic examination. The curettage is of both diagnostic and therapeutic value. Endometrium in the secretory phase signifies that ovulation has occurred. If the curettage has been performed during the third week of the cycle and shows a proliferative phase only, this is interpreted by many clinicians as signifying that the bleeding is due to an anovulatory cycle.

If the patient has reached thirty-five years of age and has completed her childbearing, x-ray therapy may be given to the ovaries at any time within a period of six months following the curettage, should excessive bleeding recur. Preclimacteric bleeding, if severe, demands curettage, and may be followed at once by the intrauterine use of radium or subsequent x-rays to the ovaries.*

Postmenopausal functional bleeding is due in most cases to a tumor (granulosal cell) of the ovary. Exploratory curettage to exclude carcinoma or polyp and subsequent oöphorectomy are indicated. It should be kept in mind that occasional bleeding results from excessive doses of estrogen prescribed for the symptoms of the menopause.

In other words, as is true of the other glands of internal secretion, overfunction in the genital sphere necessitates surgical intervention in most cases.

UNDERFUNCTION

In this group I include amenorrhea, functional sterility and dysmenorrhea. The primary site of the underfunction usually appears to lie in the prepituitary. Two constitutional abnormalities are frequent in this group, namely infantilism and eunuchoidism. The sex cycle is also unfavorably influenced by obesity, malnutrition and pulmonary tuberculosis.

Amenorrhea. Primary amenorrhea signifies the non-occurrence of menstruation at the expected age (fourteen to seventeen years). Congenital malformations must be excluded. These patients are generally infantile or eunuchoid. Secondary amenorrhea is the cessation of menstruation after the menarche. I have observed the spontaneous return of menstruation after intervals of two to four or even seventeen years.

Bioassay has shown that amenorrheic women may have normal, acyclical or excessive blood and urine cycles. No satisfactory explanation of the non-occurrence of the uterine bleeding can be offered. The amenorrheic uterus does not respond by bleeding to a dose of estrogen which produces bleeding in the human castrate. From five to ten times this dose is required in amenorrhea.

If obesity, malnutrition or markedly reduced thyroid function is noted, dietary measures and thyroid therapy cure the condition. In all other cases my therapeutic efforts have failed. Neither gonadotropic factors or estrogens have helped. In my opinion the results credited to therapy are accidental and coincidental.

Functional sterility. Many of this group are also amenorrheic. I have observed conception in amenorrheic patients. Others menstruate regularly and appear to possess normal genital organs. I have no data proving that anovulatory menstruation is more frequent in these patients than in normal women.

What has been said concerning the obese and hypothyroid in connection with amenorrhea applies likewise to the functionally sterile. Unless marked stigmas of infantilism or eunuchoidism are manifest, a prognosis is largely guesswork. My predictions have proved wrong so frequently that I no longer venture any. A single ovulation, at times followed by impregnation, has been induced by the intravenous injection of pregnant mare's serum, but I have not attempted this procedure.

Dysmenorrhea. Menstrual pain is a symptom found in normal, infantile and overworked women. It is not invariably cured by childbirth. The underlying cause is unknown. In mild cases antispasmodics such as atropine (1/250 to 1/100 gr.) combined with coal-tar drugs and codeine afford

*Radiotherapy, both with x-rays and with radium, should be given solely by fully qualified specialists.

relief. Emmenin (sodium pregnandiol glucuronidate), an estrogen extracted from the placenta, taken over a period of months, may help. In the severest grades 1 mg. of progesterone, the corpus-luteum hormone, injected subcutaneously two or three days before the expected period, and 5 mg. with the onset of the pain, relieve but do not cure.

Morphine is contraindicated. I have never performed presacral sympathectomy for this trouble. In several persistent sufferers over thirty-five years of age I have induced the menopause by means of x-ray.

PREGNANCY

During pregnancy the hormonal conditions are abruptly elevated to high levels. In the blood the gonadotropic factor increases one thousand times, the estrogenic four times. Exact figures of the change in corpus-luteum hormone are lacking. From these findings it appears illusory to give either gonadotropic extracts or estrogens in pregnancy with any expectation of therapeutic effect. Progesterone may be prescribed in habitual abortion because it does calm uterine contractions and is essential for the embedding of the ovum. It is impossible to gauge the results obtained, particularly as we know that the placenta elaborates gonadotropic, estrogenic and progestational factors throughout the duration of gestation.

MENOPAUSE

The menopause, if physiologic, is manifested by the gradual or abrupt cessation of menstruation. It may have been induced by x-rays or radium, as well as by operative castration. If the uterus alone is removed the blood cycle continues.

In the menopause, cardiovascular symptoms — flushes, sweats, palpitation and dizziness — most commonly develop. Their intensity varies in different individuals. Digestive and arthritic disturbances are frequent. Psychical and mental upsets may occur. Local genital atrophies are the rule. All these manifestations are relieved by adequate doses of estrogens given by hypodermic injection or by mouth. Some patients respond to 5000 international units given three times a week for two weeks. The treatment must be resumed when symptoms recur, if possible with progressively increasing pauses.

Objective guides of the effectiveness of the therapy are disappearance of excess gonadotropic factor in the urine and persistently positive (cornified cells) stained smears from the vagina.

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THE PRESENT STATUS OF THE BLOOD SEDIMENTATION RATE

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OBSERVATIONS on the rate of settling of the red blood cells constitute one of the oldest laboratory procedures known to medicine. The writings of Galen in the second century A.D. suggest that the humoral pathologists of his time laid great stress on this phenomenon, albeit their interpretation was erroneous. As humoral pathology fell into disrepute these hematological observations went into the discard until the subject was revived in 1772, by Hewson,[1] who apparently distinguished between the stratification of drawn blood due to coagulation and that due to settling of the erythrocytes. Nasse[2] in 1836 made an exhaustive inquiry into sedimentation velocity and the aggregation of the red blood corpuscles, and the parallelism between the two was later pointed out by Jones[3] in 1843.

The first scientific investigation of this phenomenon began with the epochal studies of Fahraeus[4] in 1917, which resulted from his accidental observations on the increased sedimentation velocity of the red cells in pregnancy. He recognized that this phenomenon was not specific for any particular disorder or physiological state, and his studies dealt chiefly with the physicochemical factors responsible. Stokes had already determined the velocity of movement of a spherical particle suspended in a fluid of lower specific gravity. Although this law of Stokes applies without modification only when the falling movement of the particle takes place in a fluid of infinite extension, it may be said for practical purposes that the rate of settling of the red cells is proportional to the difference between the specific gravity of the particle and that of the fluid, inversely proportional to the viscosity of the fluid and directly proportional to the square of the radius of the suspended particle. In blood sedimentation this radius is not the radius of the erythrocyte but of the red-cell aggregate, because of the phenomenon of clumping. Fahraeus showed that variations in the difference between the specific gravity of the blood and that of the suspended

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particle and in the viscosity of the plasma had little influence on the sedimentation velocity. Of more importance was the red-cell concentration, and of vast consequence was the radius of the suspended particle. Hence the significance of the size of the red-cell aggregates. This size is determined by properties of the blood plasma and appears to be proportional to the serum globulin and serum fibrinogen. Some authors fail to agree with this dictum, such as Aldred-Brown and Munro,[5] who could find no correlation in 54 cases of rheumatic disease between sedimentation rate and fibrinogen percentage or globulin percentage of plasma proteins, and who concluded that the sedimentation rate had no connection with the globulin-albumin ratio, the fibrinogen-globulin ratio or the ratio of fibrinogen plus globulin to albumin and to total proteins. Gilligan and Ernstene,[6] however, demonstrated a close relation between the erythrocyte sedimentation rate and the fibrinogen content of the plasma and concluded that, except in certain cases with liver damage, the plasma fibrinogen plays the major role in controlling the corrected sedimentation index. This theory is likewise accepted by Cutler, Park and Herr,[7] Wintrobe[8] and many others.

The erythrocyte sedimentation rate may be measured by any one of three methods. The Linzenmeyer technic measures the time required for the upper level of the sedimenting cells to fall a given distance. Westergren measures the distance which they fall in a specified period of time. In Cutler's[7] method a graph is constructed from the distance which the sedimenting cells fall per unit of time during an hour. In this way it is possible to demonstrate three phases in the phenomenon of sedimentation: (1) aggregation of the red cells with rouleaux formation, during which little drop in red-cell level occurs and the graph is almost horizontal; (2) the sedimentation phase, during which the graph slopes with varying degrees of abruptness; (3) the packing phase, wherein the rate of sedimentation decreases with a consequent return of the graph toward the horizontal.

Greisheimer, Treloar and Ryan,[9] in a study of the interrelation of these methods, concluded that the average sedimentation in one hour for normal subjects was reasonably concordant for the three methods despite wide differences in tube width, anticoagulant concentration and length of fluid column.

Wintrobe[10] has pointed out certain sources of error in performing the test by whatever method. The rate may be delayed by an excess of anticoagulant. Because cell volume may be materially influenced by the type of anticoagulant used he recommends the combination of ammonium and potassium oxalate devised by Heller and Paul,[11] in which the shrinking effect of the one salt offsets the swelling effect of the other. This appears to be as reliable as heparin and is certainly much cheaper. The bore and length of the tube must be kept within certain limits and the tube must be maintained in a vertical position if erroneous readings are to be avoided. Temperature likewise influences the rate of sedimentation, but the differences in temperature of the average laboratories will not cause significant error. More than four hours' delay in carrying out the test after withdrawal of the blood may likewise give rise to inaccuracies. For the actual technic of the test the reader is referred to any standard text on laboratory procedures or to the references.[10, 12, 17]

The greatest controversy has raged over the importance of correction for anemia, as it is well recognized that red-cell sedimentation is more rapid in blood having a subnormal concentration of erythrocytes. This is more marked in the short tubes, but Cutler contends that the use of the graph which differentiates the three phases of the phenomenon makes misleading corrections for anemia unnecessary. Wintrobe thinks that such variations in cell volume may interpose significant error in the final reading, and has constructed a logarithmic curve[10] for correction of the sedimentation rate according to the cell volume obtained by high-speed centrifugation at the completion of the test. He[8] admits, however, that a correction for anemia may occasionally obscure a pathologic sedimentation rate, and advises that both corrected and uncorrected rates be recorded. Unfortunately, he does not say which one is to be accepted. In spite of this ambiguity I have found the sedimentation test, performed and corrected according to Wintrobe, an exceedingly useful procedure provided its significance and clinical limitations be kept in mind. The uncorrected rate has hardly ever proved to be more accurate when compared with analogous tests and with the clinical picture as a whole. Most investigators agree that the increased length of the Westergren tube (200 mm.) obviates the need of correction for anemia or cell volume, as packing here has much less influence on the rate than it does in a shorter tube. Bannick[12] and Bouton[13] have reached substantially these conclusions after particularly careful study. Perhaps the greatest argument in favor of the Wintrobe method is that the subsequent centrifuging makes possible a determination of mean corpuscular volume and icteric index and a rough macroscopic estimation of platelets and leukocytes with a minimum of effort.

The range of normal physiologic variations of any test must be known before it can be relied

on for the detection or measurement of pathologic states. There appears to be a slight difference between the two sexes in the upper limits of normal erythrocyte sedimentation. A correction for this is made only in the Wintrobe method, and recently Wintrobe has advocated abandoning the double standard as unessential. Riseman and Brown[14] found a slight elevation of the rate to be a normal concomitant of old age although Miller[15] considered this insignificant. During pregnancy the sedimentation rate increases from the tenth or twelfth week, and does not become normal until the third or fourth week post partum. The slight fluctuations occurring during menstruation are not important.[8] The taking of food or exercise seems not to influence the sedimentation rate. The most disturbing and apparently the least appreciated cause of variations is due to meteorological conditions. Hoverson and Petersen[16] have shown that just as meteorologic changes can cause variations in leukocytes, fibrin, platelets, corpuscular resistance, and so forth, so they may result in daily variations in the same person's sedimentation rate amounting to 100 per cent.

In spite of these drawbacks the sedimentation test has proved of great value in clinical medicine. Over eleven hundred references in the *Quarterly Cumulative Index* during the last ten years, on which this review is based, attest the general interest in its clinical application. Almost every conceivable clinical entity has been investigated with this procedure, and there is hardly a country not represented in the avalanche of literature, which shows no sign of abating. One of the most comprehensive appraisals of its use was based on Cutler's[17] experience with 5000 patients over a six-year period. He emphasized, as had Fahraeus eleven years earlier, that the test was not specific for any particular disease. He pointed out that the mechanism was obscure and that the phenomenon depended on the amount of cellular destruction going on in the body. He listed the diseases giving an abnormal sedimentation rate as follows:

Chronic infection, such as syphilis or tuberculosis
Acute infection, such as pneumonia, septicemia or the exanthemas
Malignancy
Localized suppuration, such as pyosalpinx or mastoiditis
Acute intoxication, such as lead or arsenic poisoning
Endocrine disturbance, such as thyrotoxicosis

To these we may add, in the light of subsequent investigations:

Acute rheumatic fever
Acute arthritis, whether rheumatoid, gonorrheal or tuberculous
Syphilitic aortitis
Myocardial infarction
Pertussis (an abnormally low rate)
Allergy (an abnormally low rate in certain types)
Liver disease (an abnormally low rate with low plasma fibrinogen)
Diabetes (?)

Simple catarrhal inflammation, such as uncomplicated appendicitis or a cold, and chronic ulcerations of small extent, such as peptic ulcer, have little influence on the sedimentation rate. The following diseases do not influence the sedimentation rate: functional disorders, as for example of the circulation or gastrointestinal tract; certain nervous disorders like dementia praecox; focal infections; metabolic diseases (diabetes?); allergy (in certain types); skin diseases; simple growths, such as nevi, lipomas and fibromas; simple cysts; and chronic valvular disease of the heart (in the absence of congestive failure).

Most of these conclusions were endorsed three years later by Lesser and Goldberger[18] in a study of three thousand tests on 2000 patients over a two-and-a-half-year period. They found that the normally high rate in pneumonia increased with the onset of complications such as pleurisy or empyema. The sedimentation rate in tuberculosis ran parallel to the activity and extent of the infection. It was elevated in pregnancy and greatly so in the case of a ruptured ectopic pregnancy. They concluded that its greatest value was in the differentiation of acute and chronic salpingitis, as a normal sedimentation rate was practically a guarantee of the absence of an acute pelvic infection. In simple catarrhal appendicitis uncomplicated by abscess or peritonitis the rate was normal in contrast to acute salpingitis, acute cholecystitis and the condition referred to as "the acute surgical abdomen." In rheumatic fever the sedimentation rate was proportional to the degree of activity of the infection. This has been confirmed by Massell and Jones,[19] who concluded that the sedimentation rate and leukocyte count were of about equal value as tests of low-grade rheumatic fever. They believed, however, that the sedimentation rate was the more valuable single test, though it might be increased by upper respiratory infections or tonsillectomy. One wonders if the elevation in these cases with respiratory infections may not have been due to reactivation of the rheumatic infection itself, inasmuch as Gallagher[20] found no elevation of the rate in moderately severe colds in the course of examining several hundred adolescents. Lintz,[21] moreover, encountered no elevation of the sedimentation rate in chronic sinusitis, chronic tonsillitis or periapical dental infections. He concluded that the reduction in sedimentation rate often observed in arthritics after the removal

of one or more of these foci was due to the subsidence of the arthritic process itself.

Certain corrections and additions to Cutler's original list seem indicated in the light of subsequent studies. The elevated sedimentation rate in malignancy seems to depend on whether there is tissue destruction as, for example, from necrosis of the neoplasm. Reichel[22] found the rate increased in 90 per cent of cases with malignant tumors, but the evidence is not convincing that mere malignancy can give an elevated rate in the absence of tissue destruction.

Kramer[23] found the sedimentation rate elevated in 67 per cent of 366 diabetic patients. As the elevation could not be correlated with either the duration of the disease or the level of the blood sugar, he concluded that it was due to some focal infection, though this hardly appears warranted in view of the work of Lintz.[21]

Not only in rheumatic fever is the sedimentation rate increased but also in rheumatoid, tuberculous and gonorrheal arthritis.[8] In these conditions, likewise, it appears to be a valuable guide to the severity and progress of the disorder.

Danzer[24] has pointed out that the rate is elevated in syphilitic aortitis. This has been confirmed by Wood,[25] who found it elevated also in myocardial infarction but retarded in congestive heart failure. As aortic dilatation due to hypertension or arteriosclerosis is not attended by an elevated sedimentation rate, this test may prove of considerable value in establishing a syphilitic etiology in the face of the negative blood Wassermann test occasionally reported in cases of syphilitic aortitis. Riseman and Brown[14] have confirmed Wood's findings in myocardial infarction and has also observed an elevated sedimentation rate in many cases of angina pectoris. Although the figure in these cases is lower than it is in coronary thrombosis, one questions the reliability of the test in differentiating the two conditions. Certainly the writer has been repeatedly confronted with corrected sedimentation rates several times the upper limit of normal in patients whose clinical history, temperature and serial electrocardiograms only warranted a diagnosis of angina pectoris. This phenomenon may be due to minute areas of myocardial softening in the region of the coronary spasm which are too slight to produce the other clinical and laboratory evidence demanded for a diagnosis of myocardial infarction. In true myocardial infarction, however, an elevated sedimentation rate is so constant a finding, as these investigators, as well as Shookhoff, Douglas and Rabinowitz[26, 27] have shown, that one must agree with Riseman that a persistently normal or only slightly increased sedimentation rate between the fourth and the twelfth day after an attack of substernal pain is strong evidence against infarction.

Another use of the sedimentation rate not originally recognized by Cutler may lie in the diagnosis of whooping cough. Gold and Bell[28] believe that the triad of cough, leukocytosis with relative lymphocytosis and a normal or subnormal sedimentation rate is pathognomonic of pertussis, and have found a low rate in 94 per cent of the cases which have reached the stage of coughing.

Contrary to Cutler's original conclusions, allergy may affect the sedimentation rate. Schulhof[29] states that unless the rate is masked by infection it is abnormally slow in allergic patients and in their non-allergic close relatives. Another cause of a decreased rate is liver disease which results in a diminished plasma fibrinogen.[8]

Linton[30] believed at one time that the sedimentation was rapid in patients with obstructive jaundice who had a postoperative tendency to bleed, whereas it was normal or slow in those with no such hemorrhagic tendency. Clute and Veal's[31] studies have led them to question the reliability of this test, and Burke and Weir[32] state that the sedimentation rate is of no value in predicting postoperative hemorrhage in jaundice.

It is evident that we have learned little more of the fundamental mechanism of erythrocyte sedimentation since the epochal work of Fahraeus twenty years ago, in spite of literally thousands of papers on the subject. That it is not specific for any particular disease is well established. That the underlying mechanism is obscure is apparent. That it depends on, and is commensurate with, tissue destruction, however, is well agreed. The technical pitfalls are many; the intrinsic and extrinsic factors capable of distorting the sedimentation rate are insidious. Yet the very appreciation of its shortcomings and limitations makes this oldest of laboratory tests an extremely valuable tool to the clinician.

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REPORT ON MEDICAL PROGRESS

ELECTROLYTE AND WATER BALANCE*

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THE clinician without access to a modern clinical laboratory need not necessarily feel that he is unable to take advantage of many of the chemical advances made in the treatment of disease. Although accurate chemical methods have of necessity been employed in the unraveling of certain clinical problems, the knowledge so acquired not infrequently enables the experienced physician to estimate metabolic disturbances by accurate clinical observations, and thus to reduce his dependence on information provided by the more elaborate laboratory procedures. On the other hand, there are many types of disease in which accuracy of diagnosis still depends to a large extent on chemical analyses.

It is the purpose of the present review to point out some of the frequently encountered disturbances in salt and water balances in which diagnosis and therapy are based on chemical analyses or reasoning.

The expositions of Gamble,[1] Peters[2, 3] and Peters and Van Slyke[4] have clarified our understanding of the disturbances in salt and water balances associated with pathologic conditions that are frequently of concern to clinicians. It is now clear that edema and dehydration are more closely related to the masses of body fluids than to their composition, whereas acidosis and alkalosis are dependent on the relative concentration of certain cations and anions of the body fluids. Since clinically significant dehydration and acidosis or alkalosis are often associated conditions, diagnosis and therapy require attention to the amount and composition both of the body fluids and of the repair solutions administered therapeutically.

A few years ago clinical analyses of the ionic composition of a patient's blood serum were often reported in such a variety of units that the physiological significance of abnormal concentrations was difficult to visualize. For example, in analyzing a patient's serum, total cations might have been reported as 140 cc. of N/10 base per 100 cc., chlorides as 320 mg. per 100 cc., and carbon-dioxide content as 40 vol. per cent. The correlation of values, expressed in such different units, to obtain a picture of the physiologic disturbance could not be made. Indeed, some laboratories might have reported the above serum-chloride analysis as 527 mg. of sodium chloride per 100 cc., thus converting a perfectly good analysis into a misleading or meaningless result. Now, however, the expression of all plasma electrolyte concentrations in terms of milliequivalents per liter permits a ready comparison of changes which have occurred in different constituents. The normal values for the plasma electrolytes in this nomenclature are:

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CATIONS	m.eq.	ANIONS	m.eq.
Sodium	138	Bicarbonate	26
Potassium	5	Chloride	104
Calcium	5	Proteinate	17
Magnesium	2	Sulfate	1
		Phosphate	2
Totals	150		150

As an illustration of the increase in information gained by employing these recognized clinical units, we may refer again to the serum of the patient cited above. Expressed as milliequivalents per liter the total cations=140, and of the anions, chloride=90 and bicarbonate=18. If the other anions normally present, that is proteinate, sulfate and phosphate, amount to their usual value of 20 milliequivalents, one has still only accounted for 128 of the 140 anion units which we know must be present for electrical neutrality. Clearly, then, the concentration of one or more of the anions normally present has increased or some abnormal anion is present in the plasma. Clinical observation, physiological reasoning and chemical analyses, singly or together, may be used to supply more specific information. But whether or not further analyses are made, the expression of such serum analyses in a single unit of concentration clarifies the clinical significance of the data. Gamble's[5] exposition of the chemical anatomy, physiology, and pathology of extracellular fluid clearly demonstrates the manner in which this unit of concentration permits a simple graphic representation of the chemical patterns of the blood plasma in pathologic conditions, which are otherwise difficult to visualize.

Dehydration

No new single laboratory procedure has been introduced which indicates the presence and degree of dehydration better than the classical physical signs of dry tongue and mucous membranes, lack of skin turgor, sunken and soft eyeballs and, in the infant, depression of the fontanelle. Occasionally, however, dehydration associated with obesity or peripheral edema may be difficult to appraise without the aid of laboratory determinations. Those commonly used to obtain confirmatory or quantitative data are:

1. Plasma volume[6, 7]
2. Specific gravity of serum[8, 9, 10] or concentration of serum proteins (refractometric,[11] Kjeldahl[12])
3. Red-blood-cell count
4. Hematocrit
5. Hemoglobin
6. Blood nonprotein nitrogen or urea nitrogen[12]

These procedures have both practical and theoretical limitations. Determination of the plasma volume, if done accurately enough to be reliable, is too time-consuming for general clinical use. The same criticism applies to the determination of the extracellular fluid volume by the sodium thiocyanate method.[13] The last four procedures may provide evidence of dehydration, if values above the norms are obtained. But many of the patients for whom estimation of the degree of dehydration is desired suffer from other disturbances which affect the values obtained by these determinations. Starvation or Bright's disease may have lowered the serum proteins so that a normal serum specific gravity or protein concentration may be obtained in the presence of dehydration. Multiple myeloma, kala-azar or a liver tumor may give elevated values for serum specific gravity and protein concentration although dehydration is not present. A lipemia or azotemia may cause a high refractometric reading which is not the result of a high serum protein and dehydration. The red-blood-cell count, hemoglobin concentration and hematocrit reading are affected by anemia or polycythemia as well as by dehydration. The nonprotein nitrogen or urea nitrogen may be elevated because of renal insufficiency as well as by the oliguria of dehydration. Or the diabetic patient who is excreting a large volume of urine may be dehydrated without appreciable retention of nitrogen.[14]

Low serum sodium and chloride concentrations may be associated with the loss of fluids by vomiting, diarrhea and ileostomy or cecostomy drainage.[15, 16] They are also frequently observed in diabetic coma,[14, 17, 18] severe chronic nephritis[19–23] and Addison's disease.[24, 25] Though they are related to the dehydration frequently present, they are not a measure of it. However, in such cases the determination of the serum concentrations of these substances may be very helpful in explaining symptoms and guiding therapy.

Control of Hydration Therapy

More important clinically than the estimation of the degree of dehydration by laboratory determinations is the accurate control of parenteral hydration therapy by data obtained from chemical analyses.[15, 23, 26] Since in dehydration the diminished blood volume and blood flow through the kidneys usually result in oliguria and poor renal function, plasma concentration changes are prone to occur prior to an improvement in renal function which is adequate enough to defend normal plasma concentrations. Because parenteral fluid is usually required when starvation has diminished the body nitrogen and when body-fluid loss has reduced the body sodium and chloride, the administration of fluids which do

not provide sodium and chloride and plasma proteins or their nitrogenous precursors may result in a marked drop in the concentrations of these constituents of the plasma. Thus, the administration of glucose solution alone to the dehydrated patient may lower the plasma sodium and chloride concentrations to values of 115 and 60 milliequivalents per liter, respectively. Such changes so disturb the osmotic equilibrium between extracellular fluid and intracelluar fluid that loss of electrolytes from the cells or passage of water into the cells produces a marked lowering in cellular concentrations.[27–29] The administration of isotonic sodium chloride solution may so lower the plasma protein concentration that edema results.[23, 30] If pulmonary edema occurs, the effect of the therapy may be unfortunate. Or, as discussed below under the next heading, the administration of sodium chloride solution alone may produce an increase in the plasma chloride concentration and a decrease in plasma bicarbonate sufficient to produce or accentuate an acidosis. The administration of parenteral fluids may be accompanied by such a fall in plasma protein concentration that edema develops before the blood volume is restored, or by such a drop in red-cell count and hemoglobin that an anemia previously masked by the dehydration becomes apparent. In either case transfusion becomes an essential part of the hydration therapy.[31] Clearly, then, continuous parenteral therapy for periods of twenty-four hours or longer demands a knowledge of its effects on the concentrations of plasma proteins, sodium and chloride. With a properly equipped laboratory at hand, all three of these concentrations can be determined on a 1- to 2-cc. sample of serum.[10–12, 32–34] Without a laboratory at hand to guide parenteral fluid therapy, the factors described above demand due consideration.

With dehydration and starvation there occurs with the loss of extracellular water and constituents a loss of intracellular substances. This loss is not replaced by present-day parenteral fluid therapy. Therefore an essential part of repair therapy is the oral administration of food as soon as this may be given without danger of a recurrence of any gastrointestinal disturbance that may have existed. It is as yet too early to discuss the administration of amino acids parenterally. Though nitrogen balances have been maintained by such therapy, severe reactions have been encountered.[35–37]

Acidosis and Alkalosis

Diagnosis

The types of alkalosis and acidosis most frequently encountered in clinical medicine are those designated as "metabolic acidosis" and "metabolic alkalosis."[4, 38] These are the types in which hyperpnea and a low serum carbon-dioxide content accompany acidosis and in which hypopnea or Cheyne-Stokes respiration and a high serum carbon-dioxide content accompany alkalosis. Thus both the character of the respirations and a single chemical analysis indicate, in what might be termed the orthodox fashion, the nature of the disturbance. However, the diagnosis of acidosis and alkalosis cannot always be reduced to such simple indices as respiratory effort and serum carbon-dioxide content.* The respiratory center may be depressed or may be stimulated abnormally, so that the type of respiration does not reflect the disturbances in acid-base equilibrium, and a decrease or increase in serum carbon-dioxide content no longer indicates respectively acidosis or alkalosis. The Henderson-Hasselbalch equation shows clearly the relation between the variables which the clinician must consider, if he is successfully to diagnose and treat the puzzling cases. The equation states,

$$\mathrm{pH} = 6.10 + \log \frac{\cdot\mathrm{HCO_3}}{\mathrm{H_2CO_3}}$$

In normal serum, pH=7.4, the carbon dioxide from $\cdot HCO_3$=26 millimols per liter, and the carbon dioxide from H_2CO_3=1.3 millimols per liter. Keeping in mind that the concentration of H_2CO_3 in the plasma is determined by the concentration of alveolar carbon dioxide and that this latter concentration is dependent on pulmonary ventilation, it is seen that the equation provides the clinician with the essential relation between pulmonary ventilation, bicarbonate content of the plasma and plasma pH. The pH is not dependent on the total carbon-dioxide content of the plasma, which can be measured readily, but on the ratio of $\cdot HCO_3$:H_2CO_3, of which neither the numerator nor the denominator is easily measured. This leads to the difficulties which bother the clinician.

Changes in the ratio, $\cdot HCO_3$:H_2CO_3, can usually be inferred from the total serum carbon-dioxide content, the history and type of respirations. When inference of this kind is possible, the determination of the serum carbon-dioxide content permits a satisfactory estimation of the three variables in the equation. However, the puzzling aspects of many cases cannot be resolved so simply. In such cases the colorimetric determination of the serum pH,[39] in addition to the gasometric determination of total carbon-dioxide content of the serum, shows whether alkalosis or

*For the ordinary clinical laboratory the determination of the carbon-dioxide content seems preferable to the determination of the carbon-dioxide combining power of the plasma.[12]

acidosis is present and, if desired, permits the estimation of the $\cdot HCO_3$ and H_2CO_3 concentrations. For, from the original equation, since $\cdot HCO_3 = \text{total } CO_2 - H_2CO_3$,

$$H_2CO_3 = \frac{\text{total } CO_2}{1 + \text{antilog } (pH - 6.1)}$$

Shock and Hastings[40] have recently introduced a method of doing both determinations on 0.1 cc. of capillary blood.

From the Henderson-Hasselbalch equation is it clear that a low total carbon-dioxide content of serum, that is the carbon dioxide from both the $\cdot HCO_3$ and the H_2CO_3, indicates a low pH and acidosis only if the ratio, $\cdot HCO_3 : H_2CO_3$ is less than normal. Such a lowering of both total carbon dioxide and the ratio occurs where the primary change is a reduction of the $\cdot HCO_3$ either by loss of sodium or by accumulation of anions other than $\cdot HCO_3$ in the blood or by both. This is by far the commonest type of acidosis and has been termed *metabolic acidosis*. It occurs in diabetic acidosis, the acidosis of severe renal insufficiency and the acidoses which accompany severe exercise, the ingestion of acids or acidifying salts and dehydration due to loss of gastrointestinal secretions and to the ensuing oliguria.

From the equation it is also clear that a low total carbon-dioxide content of serum may be associated with an increase in the $\cdot HCO_3 : H_2CO_3$ ratio above normal and therefore with an increase in pH and alkalosis. This situation occurs where the primary change is a decrease in alveolar carbon-dioxide pressure which causes a decrease in carbon dioxide dissolved in the plasma and a decreased carbonic acid (H_2CO_3) concentration. This has been called the alkalosis of primary carbonic-acid deficit[4] or more simply, *respiratory alkalosis*.[38] It may be the result of the hyperpnea of encephalitis,[41] hysteria,[42] certain respiratory stimulants and oxygen lack.[43]

Again referring to the equation, a high serum carbon-dioxide content may be associated with an increase in the ratio and alkalosis. This has been called *metabolic alkalosis,* and may occur with the ingestion of alkaline salts, particularly in the presence of renal insufficiency[44] or with the loss of chloride in excess of sodium as frequently results in gastric vomiting.[45]

Or a high serum carbon-dioxide content may be associated with a decrease in the ratio and an acidosis. In this case the acidosis is one of primary carbon-dioxide excess[4] or a *respiratory acidosis*.[38] It may occur where there is suppression of the activity of the respiratory center because of toxins or exhaustion, paralysis of the respiratory muscles, upper respiratory obstruction or emphysema.

An accurate diagnosis of the type of alkalosis or acidosis is a prerequisite to the best therapy. The dependence of the clinician on laboratory analyses will vary with individual circumstances.

For example, in a case of diabetic coma, where the history is typical, the physical signs of dehydration, ketosis and hyperpnea are classical, and the glucosuria, ketonuria and hyperglycemia confirm the diagnosis, the chemical changes in the blood conform so closely to the pattern established by previous studies[14, 17, 18, 46] that the quantitative determination of the degree of acidosis contributes little diagnostically or therapeutically unless complications arise.

On the other hand, in a dehydrated patient with a history of diarrhea and vomiting, where the respiratory response may be depressed by toxemia and the urinary findings will be of almost no assistance,[47] the differential diagnosis of metabolic acidosis and metabolic alkalosis may depend on the determination of the carbon-dioxide content of the serum. Not infrequently a desirable understanding of the extent of specific concentration changes in the plasma occasioned by the loss of fluids is obtained only after determining the serum sodium and chloride concentrations as well.

In the treatment of a nephritic patient with acidosis it is important to know whether the lowered serum carbon-dioxide content is due to a diminished serum sodium or to an increase in the concentration of serum anions caused by the retention of metabolites. And the recognition of the recently described syndrome of chloride acidosis and nephrocalcinosis[48, 49] depends largely on the detection of a lowered serum carbon-dioxide and an elevated serum chloride concentration.

The diagnosis of respiratory alkalosis or acidosis usually depends on the determination of plasma pH and carbon-dioxide content. The determination of serum sodium and chloride concentrations may be helpful.

Finally, there are cases where combinations of conditions make appraisal of the situation extremely difficult. A case of methyl salicylate poisoning,[50, 51] for example, may be very puzzling. The vomiting tends to reduce the plasma chloride and to produce a high serum carbon-dioxide content and alkalosis. The salicylate, as a foreign organic anion, may tend to lower the serum carbon dioxide and produce an acidosis. The drug may stimulate the respiratory center, producing a hyperpnea with a low serum carbon dioxide and respiratory alkalosis. A correct appraisal of the situation, in order to ascertain whether sodium lactate or bicarbonate is indicated or definitely contraindicated by the hyperpnea, may require careful integration of the history and physical findings and several chemical determinations.

Shock and Hastings[38] have pointed out that the application of microtechnics in determining the acid-base equilibrium of the blood permits studying the paths of acid-base displacement and recovery after experimental displacement. Their use of triaxial co-ordinates is particularly appropriate for the graphic presentation of the three variables of the Henderson-Hasselbalch equation. Moreover, by plotting the logarithm of the percentage change in bicarbonate against time they obtained a linear relation in which the slope, "K," characterized the rate at which the blood returned from the experimental displacement to normal. Because the re-establishment of equilibrium depends largely on renal function their "constant of elimination" should prove useful in the evaluation of renal disease.

Therapy

Clinically, *respiratory acidosis* is not in itself serious. In many cases the increase in plasma carbonic acid is beneficial because it stimulates respiration. Treatment, therefore, is directed not toward the acidosis but toward the cause underlying the respiratory disturbance.

Respiratory alkalosis only infrequently leads to symptoms of tetany which require treatment per se. If the tetany does demand immediate specific treatment, air containing an increased amount of carbon dioxide may be administrated by having the patient rebreathe from a bag or from a closed oxygen tent with the soda lime removed from the system, or by supplying carbon dioxide directly to the inspired air. Morphine may also be helpful. The administration of calcium or ammonium chloride by mouth may increase the compensatory elevation of plasma chloride. Permanent relief, however, depends on treatment of the underlying disturbance. To mistake this type of alkalosis for acidosis and to administer an alkaline sodium salt may lead to serious tetany.

Adequate treatment of *metabolic alkalosis* resulting from an intake of alkaline sodium or potassium salts which exceeds the kidney's excretory ability is usually accomplished by discontinuing the alkaline salt and administering such amount of fluid as will provide a large volume of urine.

Metabolic alkalosis caused by the loss of gastric hydrochloric acid usually occurs in patients whose vomiting prevents the oral ingestion of food, fluid or medication. Dehydration and some degree of starvation therefore accompany the alkalosis and also demand treatment. Parenteral physiological saline solution is ideal for correcting the alkalosis and dehydration. It provides the three essential substances, water, sodium and chloride, and in supplying the two latter in equal amounts* tends to increase the plasma chloride concentration and to diminish the alkaline reserve.[52] The intravenous infusion of 10 per cent glucose solution stimulates the circulation, treats the starvation and provides fluid for an adequate volume of urine.

Metabolic acidosis due to sodium deficit sustained through loss of body fluid is usually accompanied by a gastrointestinal condition that prevents either the ingestion or absorption of substances taken orally. Dehydration and starvation are almost always present and demand treatment. The therapy must usually be provided by intravenous infusions and clyses or by continuous intravenous drip.

The dehydration and acidosis are treated by the parenteral administration of fluid containing chloride and sodium in concentrations appropriate to repair the plasma and extracellular fluid deficits and to correct the acidosis by providing for an increase in the diminished alkaline reserve. In the presence of the diminished renal function it is clear that physiological saline solution* is not an appropriate solution for the initial parenteral therapy. In the dehydrated oliguric patient physiological saline solution tends to raise the serum chloride, lower the alkaline reserve and lower the pH of the blood.[15, 26] The introduction of sodium *r*-lactate for parenteral therapy by Hartmann and Senn[53, 54] provides a convenient, economical and appropriate parenteral solution. One part of M/6 sodium *r*-lactate solution to two parts of physiological saline solution makes a solution which contains approximately 150 milliequivalents of sodium and 100 milliequivalents of chloride per liter. The administration of this solution to the dehydrated acidotic patient provides the means of repairing the dehydration and restoring the alkaline reserve. If the acidosis is extreme, equal parts of the two solutions may be used in the initial stages of therapy. The total amount of solution to be administered is determined by the amount required to correct the dehydration. The rate of administration is based on the amount that can be given safely to each individual patient per unit of time. The therapy is controlled as already discussed under the section on the control of hydration therapy. The specification of a given number of cubic centimeters of M/6 sodium lactate solution and physiological saline solution per kilogram of body weight[55] seems too rigid and dogmatic to be recommended. The administration of

*The concentrations of chloride and sodium in physiological saline solutions are each approximately 155 milliequivalents per liter. Hence the chloride concentration exceeds the normal plasma chloride concentration by 50 milliequivalents per liter.

that amount of sodium which by calculation[54, 56] should bring the alkaline reserve, as measured by the serum carbon-dioxide content, back to normal seems theoretically of doubtful validity and practically an unphysiological procedure. There seems little justification to the use of solutions which substitute expensive Ringer's solutions for physiological saline solution. If calcium is needed, its specific administration in therapeutically effective amounts seems preferable to relying on the calcium supplied in Ringer's solution. There is no evidence to support the parenteral administration of potassium.

Except in cases of diabetic coma where intravenous glucose is not helpful and may be harmful,[57] the starvation is treated by 10 per cent glucose given intravenously. If the starvation is a prominent part of the picture, the basic caloric needs can be met by glucose supplied by a continuous intravenous drip without entailing hyperglycemia and glycosuria.

Metabolic acidosis due to retention of anions occurs as the result of renal insufficiency, faulty metabolism or the administration of salts which demand the differential excretion of so-called "fixed base." The last type of retention is usually corrected when the administration of the salt is discontinued. Diabetes mellitus and starvation both result in the retention of the ketone acids. The use of glucose in the treatment of the ketonemia of starvation has already been mentioned. The ketonemia in diabetes mellitus is treated by the administration of insulin while the dehydration and acidosis due to sodium deficit are being treated as outlined above. The retention of anions due to renal disease is frequently associated with diminished plasma sodium and chloride concentrations.[19–23] Treatment consists in providing a diet that demands a minimal excretion of the retained catabolic products, an adequate caloric intake, a liberal sodium and chloride intake (the former being somewhat in excess of the latter) and a large volume of urine.[19, 58, 59]

The recent work of Guest and Rapoport[60, 61] on the role of diphosphoglyceric acid in the acid-base equilibrium of the blood cells has important therapeutic implications. Phosphate appears to be an essential factor in the restoration of the cells' normal chemical structure. If effective methods of providing phosphate can be found, the treatment of acidosis may be extended beyond the confines of the inactive plasma and extracellular fluids and into the regions of active cellular metabolism.

Addison's Disease

Loeb[24] and Loeb, Atchley and Parson[25] have called attention to and carefully analyzed the abnormal pattern of the blood serum in Addison's disease. The abnormalities commonly found are a decrease in serum sodium and glucose concentrations and an increase in the concentrations of serum potassium and nonprotein nitrogen.

The marked increase in the urinary excretion of sodium and chloride in Addisonian crises and the restoration of normal excretion by use of cortical extracts have been described by several groups of workers.[62, 64]

The beneficial effects of the ingestion of sodium chloride in such amounts as will maintain normal plasma concentrations and salt balance have been demonstrated by Loeb and his co-workers,[65, 66] Harrop, Soffer, Nicholson and Strauss[67] and others. The desirability of a restricted potassium intake as recommended by Wilder, Snell, Kepler, Rynearson, Adams and Kendall[68] has not been confirmed by general experience.

Loeb, Atchley, and Parson[25] concluded that a decrease in serum sodium concentration was the most sensitive chemical criterion of adrenal insufficiency in man. Harrop and his co-workers[63] have suggested the use of a salt-free diet as a diagnostic test. During the period of salt restriction, which is usually from three to five days, the patient is observed for symptoms of an Addisonian crisis, for a decrease in plasma sodium and chloride and for a high urinary chloride excretion. Because the test may provoke a serious crisis, they call attention to the necessity of the closest medical supervision. Zwemer and Truszkowski[69] suggested the diagnostic procedure of feeding 10 to 20 mg. of potassium per pound of body weight and determining the concentration of potassium in the serum at frequent intervals. Wilder and co-workers[68] suggested the ingestion of potassium as a means of provoking an increased excretion of sodium and potassium.

However, chemical studies to determine serum concentrations and urinary excretions under the controlled conditions mentioned above provide confirmatory, not specific, evidence for the diagnosis of Addison's disease, and entail appreciable risk to the patient. The changes in the chemical composition of serum and the negative sodium and chloride balances which occur in Addison's disease are frequently observed in nephritis,[19–23] asthenia,[70] dehydration[15, 16, 71] and uncontrolled diabetes.[14, 17, 18] The ingestion of potassium salts by the nephritic patient may be associated not only with an increase in plasma potassium and urinary sodium excretion but also with serious untoward symptoms.[72–76]

More recently Cutler, Power, and Wilder[77] have described a simplified procedure for the diagnosis of Addison's disease. The test involves the administration of standard amounts of water and potassium and sodium chloride for two days and the determination of the concentration of sodium or chloride in the urine the morning of the third day. Because the analysis of chloride in urine is easier than that of sodium, chloride alone usually is determined. Urinary concentrations of chloride greater than 63 milliequivalents per liter are stated to indicate adrenocortical deficiency. The specificity of this test still needs verification by demonstrating its ability to distinguish Addisonian patients from those with the conditions mentioned above who tend to excrete large amounts of sodium and chloride in the urine, even in the presence of low sodium and chloride concentrations.

Moreover, the repeated observation of Addisonian crises with hemoconcentration and diminished plasma volume, without a decrease in the serum sodium concentration and without a loss of body sodium, chloride or water by way of the urine, suggests the important role that abnormal distribution of water and electrolytes may play in this disease.[25, 78–80] Data are not yet available which are adequate to show that the determination of the total plasma sodium (as measured by the plasma volume and sodium concentration) is a more satisfactory criterion of Addison's disease than are serum concentration and urinary excretion values.

The recent work of Thorn, Engel and Eisenberg[81] with desoxy-corticosterone gives promise that this synthetic product will provide more economical and effective therapy than any now available.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 25201

PRESENTATION OF CASE

A twenty-seven-year-old single male office worker was admitted complaining of loose bowel movements of one and a half years' duration.

Vague gas pains, coming on usually about 3 p. m. and relieved by changing position or massaging the abdomen, had been noted about one and a half years prior to admission. They were felt in the right upper quadrant over an area the size of the patient's hand. He then began to lose weight gradually in spite of a good appetite and an adequate diet. He gradually developed loose stools which contained mucus and, ten months before entry on two separate occasions, blood streaks. He had noticed none of these since that time. Six months before admission he was having four or five loose movements a day and occasionally one at night. These continued until two months before entry at which time they decreased to one or two movements a day though still associated with mucus. There was no significant pain. He had eaten fruits, vegetables and meat but avoided milk because it seemed to increase the mucus.

His past and family histories were noncontributory.

Physical examination showed a markedly undernourished man who appeared to be slightly anemic. Examination of the head and chest was negative. The blood pressure was 110 systolic, 70 diastolic. There was slight tenderness above and to the right of the umbilicus.

The temperature was 97.4°F., the pulse 120, and the respirations 20.

Examination of the urine was negative. The blood showed a red-cell count of 3,270,000 with 60 per cent hemoglobin, and a white-cell count of 12,400 with 85 per cent polymorphonuclears. The serum protein was 6.4 gm. per 100 cc. A blood Hinton test was negative. The sedimentation rate was 18 mm. in fifteen minutes, 40 mm. in thirty minutes, 50 mm. in forty-five minutes, 56 mm. in sixty minutes. Numerous stool examinations were negative for blood and for parasites.

X-ray films of the chest were negative. The upper gastrointestinal tract was normal. The barium passed through the small bowel rapidly. The terminal ileum showed abnormal mucosa, appearing nodular proximally and destroyed in the region of the ileocecal valve. There was no "string sign," the ileum being of normal caliber. The ileocecal valve was elongated and irregular. A barium enema showed that the colon was grossly abnormal. The ascending colon showed numerous rounded and irregular filling defects; in places these simulated polyps, while in other areas the defects were plateau-like and larger. A flat mass measuring up to 3 cm. in diameter was seen just proximal to the hepatic flexure. The margins of the ascending colon showed fine, irregular projections simulating ulcerations. These apparent ulcerations were spotty in distribution, some of them being seen in the descending colon as far as the crest of the ileum, whereas parts of the transverse colon were fairly normal. The sigmoid and rectum were essentially normal.

On the third hospital day proctoscopy was done. The mucosa was granular and injected but not thickened or edematous. There were no ulcerations or hemorrhagic areas.

Transfusions, vitamins and a high caloric diet brought about little improvement. On the fifty-third hospital day an operation was performed.

X-RAY INTERPRETATION

DR. AUBREY O. HAMPTON: This was an interesting case. I did the examination, and I was particularly impressed by the location of the changes in the colon and the size of the polypoid defects. Then there was a segment in the transverse colon which was fairly normal, and another area of smaller ulcerations at the splenic flexure and at the beginning of the descending colon which faded out toward the sigmoid. These polypoid-appearing lesions are quite definite and involve the whole ascending colon and hepatic flexure. In some films taken during fluoroscopy with pressure on the lesion there seemed to be one solid plateau or one elevation separate from the polyps. I think it is possible that pressure caused multiple polyps to blend into one another and to produce a defect similar to a flat mass. You can see there that the surface is granular. The colon is small in the proximal portion but of normal caliber from there on. The ileum shows an area of disease near the valve, but even there it is not constricted. The valve is elongated and irregular. The small bowel is normal. The chest was normal.

DIFFERENTIAL DIAGNOSIS

DR. E. PARKER HAYDEN: This story is more suggestive of an inflammatory process than it is of malignant disease. The laboratory findings were essentially negative except for a slight leukocytosis and a somewhat rapid sedimentation rate

which suggest infection. The stools were negative for blood, parasites and amebae.

The gastrointestinal x-ray findings are quite clear and yet it is difficult to make a definite diagnosis from them. The chest films were negative; while such a finding suggests that the intestinal condition was not due to tuberculosis, this is not necessarily so. The lack of a "string sign" in the ileum does not rule out regional ileitis because this is simply an indication of a very narrow ileum with marked spasm. Not all cases of regional ileitis exhibit this picture. The defects which appear in the x-ray resemble real polyps rather than the pseudopolyps which occur in ulcerative colitis and represent islands of ragged mucosa in the midst of areas of destruction. On the other hand, some cases of ulcerative colitis develop true adenomatous polyps.

We hear more and more about resections in regional ulcerative colitis which can occasionally be done when the lesions involve only the right colon. This patient was proctoscoped, and a note made that the mucosa was injected and granular but not thickened and edematous. I cannot accept this statement as correct because if the mucosa is granular I believe that it is always thickened. If the mucous membrane of the rectum were really granular, I should consider it diagnostic of mild ulcerative colitis. It is a question of the interpretation of that particular proctoscopy. I believe there is not much doubt that this patient had an ulcerative process which involved largely the right colon and the ileum. It has been thought that there may be a relation between ulcerative colitis and regional ileitis. One patient whom I had previously proctoscoped and on whom I had made a definite diagnosis of ulcerative colitis was operated on subsequently by another surgeon who resected two isolated areas of what he considered to be typical regional ileitis.

There is one other possible diagnosis to be considered — multiple polyposis. In this disease, although diarrhea accompanies it, we ordinarily do not see inflammatory changes of any extent in the mucosa. Furthermore, polyps are almost always present in the rectum. There were none in this case. Multiple polyposis is a familial disease and does not fit the picture in this case.

I believe the most likely diagnosis is an ulcerative inflammatory process of the right colon, of nonspecific rather than tuberculous origin, with secondary polyposis.

Dr. Chester M. Jones: When this young man came into the hospital the story was very suggestive of a partially obstructive process in the region of the terminal ileum, and before we heard the results of x-ray study, I wondered whether he did not have terminal ileitis or tuberculosis of the ileocecal region. Proctoscopy showed just about what was described in the protocol, but I should have added the note that the rectosigmoid looked more as if it were irritated secondarily from some process higher up than as if there were primary disease in that area. I think occasionally one gets a confusing picture of irritation and even partial superficial erosion which is not due to ulcerative colitis. The x-ray films certainly showed trouble in the region of the ileocecal valve but much more in the colon than I anticipated, and before operation it seemed to me that there was still a possibility of a tuberculous process. The obvious thing to my mind was that this boy was terribly undernourished. We decided that as soon as we got him in decent shape he would have to have an ileostomy and a resection of a large part of the bowel, how much, only to be determined at operation. If I remember correctly, when I went over the films with Dr. Hampton we agreed that there was involvement of the upper portion of the ascending segment of the colon but that there was also something in the region below the splenic flexure.

Dr. Arthur W. Allen: Before operation I thought of all the possibilities that Dr. Hayden has mentioned, and we did put a good deal of stress on the splenic flexure. You may have forgotten it, Dr. Hampton, but we found areas in this region that were definitely diseased. At exploration it was very interesting that the transverse colon was as normal as any large bowel I have ever seen. His ascending colon was diseased and his descending colon was involved almost to the rectosigmoid junction. There was this definite skip of the transverse colon. So far as you could tell grossly the involved bowel could have been diseased by any one of the processes which Dr. Hayden has mentioned. Inasmuch as the rectum had appeared quite normal by proctoscopic examination and also seemed fairly normal to palpation, the ileum was transected and the proximal end anastomosed to the rectosigmoid. He did reasonably well aside from an ischiorectal abscess, but did not gain so much as we thought he might. After removing his right colon, at a second operation, he picked up fairly rapidly and gained as much as a pound a day, so that he was in a practically normal state of nutrition at the time of discharge. In view of the definite lesions in the descending colon it is difficult to explain why he has done so well; perhaps this is due to better drainage of this segment, or simply to less disease.

Clinical Diagnosis

Regional ileitis?
Ulcerative colitis?

Dr. Hayden's Diagnosis

Non-specific ulcerative ileocolitis.

Anatomical Diagnoses

Ulcerative colitis.
Adenomatous polyps?

Pathological Discussion

Dr. Tracy B. Mallory: The specimen showed all the usual features of a typical ulcerative colitis and, in addition, a marked polypoid hypertrophy of the remaining mucosa, in fact more marked than any we have ever seen. Some of the polyps were large enough, I think, to be considered adenomatous polyps. Between them there were characteristic areas of inflammatory thickening of the mucosa, of ulceration and of scarring of the muscular and serosal layers. There was nothing to suggest carcinoma.

Dr. Hayden: What was the flat mass 3 cm. in diameter described in the x-ray — a cluster of polyps?

Dr. Mallory: I should think that was it. We found nothing else that would explain it.

Dr. Allen: Do you want to say how many cases of ulcerative colitis of this degree you have seen without involvement of the rectum?

Dr. Mallory: Very few if any.

Dr. Allen: How about you, Dr. McKittrick?

Dr. Leland S. McKittrick: I have never seen one.

Dr. Allen: Dr. Jones, is it new to you?

Dr. Jones: Yes.

Dr. Allen: And you, Dr. Urmy?

Dr. Thomas V. Urmy: I have never seen it.

Dr. Allen: That is why I thought it must be something else and why I connected the rectosigmoid with the terminal ileum. I have seen only one other patient that had had it done, and his rectum finally had to be removed.

Dr. Hampton: Certainly it is an unusual x-ray appearance. I do not believe you could get one out of a hundred to say that the lesion was due to ulcerative colitis.

CASE 25202

Presentation of Case

A fifty-six-year-old married Italian barber was admitted complaining of an abdominal mass of six months' duration.

Twenty-two years prior to entry the patient was told that he had a right kidney stone, the diagnosis having been established by x-ray examination. Six months before admission he first noted a small mass high in his right upper quadrant, beneath the costal margin. It was non-tender and caused no symptoms. Six weeks before entry he first noticed that his urine contained blood. He continued having hematuria intermittently with no urinary symptoms except occasional frequency. Three weeks later he noted that the mass was the size of an orange. It continued to grow until it occupied most of his right upper and lower quadrants. It was not tender, was movable and caused a dragging sensation while standing. His hematuria continued but was his only urinary complaint. He had been unable to eat anything but milk and eggs during the previous few weeks and had lost 20 pounds in weight during the two months before entry. He had had no other gastrointestinal symptoms, having had regular normal bowel movements and no evidence of blood in the stools. His past and family histories were noncontributory.

Physical examination showed a tall, nervous man weighing 200 pounds. Examination of the chest was negative. The blood pressure was 150 systolic, 95 diastolic. The abdomen contained a large soft mass the size of a football extending from the right upper quadrant down past the iliac crest. It was freely movable, non-tender and smooth, and descended with respiration.

The temperature was 99.5°F., the pulse 80, and the respirations 20.

The urine examination showed a specific gravity of 1.025, a trace of albumin, no casts, an occasional white blood cell and numerous red cells. Culture showed a moderate growth of *Staphylococcus aureus*. The blood showed a red-cell count of 5,000,000 with 100 per cent hemoglobin, and a white-cell count of 8000. The nonprotein nitrogen of the serum was 26 mg., the chloride 97 milliequivalents, the carbon-dioxide combining power 55.3 vol., the protein 6.4 gm. and the sugar 97 mg. per 100 cc. A blood Hinton test was negative. The serum calcium was 8.92 mg. per 100 cc., the phosphorus 3.64 mg.

X-ray films of the chest and skull were negative. A flat abdominal film showed a large mass in the right abdomen extending from above the costal border to the crest of the ilium. No kidney shadow was visible outside of this mass. There was an ovoid area of calcification 2.5 by 1.5 cm. overlying the right side of the sacrum. The right half of the pelvis, the right femur and the fourth lumbar vertebra showed evidence of extensive Paget's disease. An intravenous pyelogram showed prompt excretion of the dye on the left, filling a nor-

mal kidney pelvis and ureter. No excretion was visible on the right side after thirty minutes. After injection of the lower part of the right ureter through a catheter, the tip of the catheter lay just above the ureterovesicular orifice. The injected material passed upward to the area of calcification and showed the lower ureter to be grossly abnormal. Multiple irregular filling defects were demonstrated in the dilated ureter between the area of calcification and the ureterovesicular orifice.

On the fourth hospital day an operation was performed.

Differential Diagnosis

Dr. Fletcher H. Colby: The significant points in the patient's history are the diagnosis of right renal stone twenty-two years previously, the patient's observation of a tumor in the right upper quadrant six months before admission, and the fact that this tumor had increased rapidly in size during the past six weeks. Hematuria had been present for at least six weeks. The absence of severe pain is also a feature of the history. Physical and x-ray examinations seem to localize this mass to the right kidney, since no function could be demonstrated by intravenous pyelogram and no kidney outline was visible by x-ray. Hematuria in the majority of instances is due to tumor or tuberculosis. Other causes are inflammatory lesions, polycystic kidneys and certain systemic conditions such as purpura. I believe that the uncommoner causes of bleeding can be excluded by the normal laboratory findings and the x-ray examination. Tuberculosis does not merit serious consideration because of the noncontributory past and family histories and the absence of a marked degree of bladder irritability.

A renal stone was demonstrated by x-ray twenty-two years before, and the findings at entry were those of stone in the right ureter with hydronephrosis. The most reasonable explanation of the rapid growth of this tumor is the development of hydronephrosis. The diagnosis of stone and hydronephrosis would satisfactorily account for the symptoms and the findings in this patient with the exception of the x-ray appearance of the lower portion of the right ureter. These filling defects cannot be disregarded. They suggest papillary tumor, which is either primary in the ureter itself or possibly has grown down from the kidney pelvis and involved the ureter. A primary tumor of the ureter is a very unusual condition, but I do not see why it could not be present in this patient. I cannot go farther in the way of diagnosis without seeing the x-ray films.

In this film there is a large area of calcification in the right ureter. I assume the bone changes are those of Paget's disease and not due to metastatic carcinoma, although I do not know how to tell the difference. My diagnosis is primary carcinoma of the ureter.

Clinical Discussion

Dr. George G. Smith: I do not remember whether we suspected a ureteral carcinoma when we operated on this patient. I think we did not, because I operated on him, and as I remember it, I was somewhat surprised at the findings. I went in to do a nephrectomy and found a big hydronephrotic kidney which I took out. The ureter was dilated down to the pelvic brim. We passed a uterine sound down the ureter to find out where the stone was, and ran into an obstruction. I palpated the ureter and found a solid mass filling the ureter from a point just above the pelvic brim to one down over the brim. At that time I realized that we had a carcinoma of the ureter; so I tied the upper end of the ureter, closed the kidney wound, put the patient on his back and made a midline incision, stripping the peritoneum inward. I picked up the ureter, which was the size of my thumb; it was easily freed down to the bladder, as is so often the case with these tumors. Since it seemed to be normal for several centimeters above the point of entrance into the bladder, I did not make an elliptical incision including the entire right ureteral ridge but cut off the ureter just at the bladder wall and removed the lower portion of it.

This patient came in yesterday to the Tumor Clinic. He looks fine and feels perfectly well. I think he must have Paget's disease in the bones because he had no complaints. I asked him if he had bled, and he said that two weeks ago he had seen a few blood flecks in the urine. We cystoscoped him, and up in the dome of the bladder we found two small papillary tumors. I could feel no mass in the operative region. On bimanual rectal examination there seemed to be no thickening in the right side of the pelvis, but of course these cases of primary carcinoma of the ureter that get well are few.

Clinical Diagnoses

Right ureteral stone.
Hydronephrosis.

Dr. Colby's Diagnoses

Primary carcinoma of the ureter.
Hydronephrosis.
Ureteral calculus.

Anatomical Diagnoses

Primary papillary carcinoma of the ureter.
Ureterolithiasis.
Hydronephrosis.

Pathological Discussion

Dr. Tracy B. Mallory: The lumen of the ureter was greatly dilated around a stone, and the stone itself was completely embedded in shaggy papillary masses of neoplastic epithelium. There was no gross or microscopic invasion of the wall that we could make out. The kidney lesion was a simple hydronephrosis, and there was no evidence of papillary tumor in the pelvis, so that there is no doubt in this case that the tumor was primary in the ureter. The later appearance of papillomas in the bladder, way up in the dome and nowhere near the mouth of the ureter, suggests, as do so many of these cases, that one is dealing with an underlying disease of the transitional epithelium that lines the entire urinary tract. This is evidenced by a tendency to the formation of papillary neoplasms throughout the tract, and in many cases the reappearance of tumor has to be looked on as a new primary tumor and not metastasis. In this case I should think that that was almost certainly true.

How would you feel about that, Dr. Smith?

Dr. Smith: The point against that is the fact that there are no papillomas in the other kidney and yet they are present in the bladder. I agree with you that it does seem as if there were some predisposing condition of the bladder mucosa that makes these tumors develop. That is a line of argument I have used regarding many bladder carcinomas, but I do not know that it applies in this case. I could not believe that it was due to a bit of tumor being borne down the ureter and being implanted, but that is possibly the case.

Dr. Mallory: Primary carcinoma of the kidney pelvis or ureter is an extremely rare disease, and on a statistical basis, one would not expect many patients to have it in both kidneys.

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ANNUAL MEETING

This issue of the *Journal* presents the complete program of the one hundred and fifty-eighth annual meeting of the Massachusetts Medical Society, to be held in Worcester at the Municipal Memorial Auditorium, Tuesday, Wednesday and Thursday, June 6, 7 and 8. The Auditorium is ideally suited for the meeting. Surrounded by adequate parking facilities, it has one large hall to house the scientific and commercial exhibits, a theater in which to hold the consecutively run section meetings, rooms for discussion groups and committee meetings, and space available for a cafeteria where lunches will be served; — all the daytime program will be carried out under one roof. The evenings' events, the Shattuck Lecture and the annual dinner, will be held at the Hotel Bancroft.

The changed form of this year's program for the section meetings should receive the attention of the fellows of the society, and it is hoped, their approval. All the section meetings are to be held in the Little Theater of the Auditorium. Unlike former years, these meetings are to be consecutive general assemblies with no two sections holding simultaneous meetings. Not only does such an arrangement promise a large attendance at the individual section meetings, but the fellows will be enabled to attend any or all the meetings without the necessity of missing one of two topics in which they are interested, an inevitable occurrence while more than one of the sections were meeting simultaneously as under the form of program previously followed.

In 1937 a day devoted to a combined clinical meeting was introduced to our program. Its success both then and last year was definite. This year a somewhat different plan has been developed by the Worcester committee for Wednesday, June 7. The morning is to be taken up by a combined meeting at which Dr. Richard B. Cattell, of Boston, Dr. Arthur M. Fishberg, of New York City, Dr. Walter C. Alvarez, of Rochester, Minnesota, and Dr. Emil Novak, of Baltimore, will present subjects of interest and importance to physicians regardless of whether they are engaged in the practice of general medicine or of the specialties. Wednesday afternoon is to be devoted to round table discussions. Three of these will be run simultaneously with a change of subject every hour. The nine subjects to be discussed are: "Craniocerebral Injuries," "Heart Disease," "Hyperthyroidism," "Pneumonia," "Low-Back Pain," "Peripheral Vascular Disease," "Urinary Infections," "Neonatal Injuries" and "Adolescent Prepsychotic Conditions."

The entire program of this year's annual meeting has been worked out thoughtfully with one chief objective: to present to the fellows of the Society through the medium of speakers of wide experience in their respective fields more or less specialized subject matter of general importance.

SPEAKING OF BATH TUBS

A TREATISE could be written on the impact of plumbing on American life and morals, to say nothing of our art and letters. Not too many years ago this influence was practically nil; the lead pipe and the tin tub were viewed askance by the virile majority of our ancestry, at a time when the flush toilet was considered in many quarters as little short of immoral.

As wealth accumulated and men decayed, an easier attitude was adopted toward hot and cold running water; it was conceded that cleanliness could still be a virtue, even if not attained under the barnyard pump with a bar of laundry soap and a curry comb. The plumber was accepted as a member of society and admitted to many churches in good standing, although, as a class, the knights of the Stillson wrench were still held up as models of clumsiness. As late as 1920 the likening of a surgeon to a plumber constituted fighting words, and even members of the new genitourinary specialty were inclined to be annoyed. Since the panic of 1929, anyone who can charge for his time while going back to the office after a forgotten blow torch is held in high esteem, and many an erstwhile banker has surreptitiously learned how to change a faucet washer and sweat a mean joint.

The bath tub is, of course, no innovation. Many Pompeians and Herculaneans were caught at the bath when proud Vesuvius erupted, and Gibbon practically attributed the decline and fall of the Roman empire to the prevailing use of the hot bath at that period of history. Warm baths are so relaxing!

Are we to meet a like fate? It is the thought that tortures us when we read in *The Commentator* that since 1900 more than 16,250,000 bath tubs have been manufactured and sold in this country. If cleanliness has come that close to godliness since the turn of the century, then woe betide us!

We recall (editorially speaking) a circular tin affair, about six inches in depth, that our paternal ancestor once had built for his personal use, plagiarizing on an English model. It was filled, pail after pail, by a perspiring servant, and was a far cry from the tinted pleasantries of the twittering twenties, but still, the menace was there.

There are no two ways about it. Plumbism has its insidious dangers. We (editorially speaking) are reminded of a family that kept a tame otter by the name of Josephine. Apparently otters are readily tamed, except for certain atavistic tendencies. Uninitiated guests, on asking for the privilege of washing (sic) before dinner, would be confused and a little bewildered by finding on the bathroom door a neatly lettered placard: "Please keep the door shut, as Josephine likes to sleep in the toilet."

MASSACHUSETTS MEDICAL SOCIETY

LEGISLATIVE NOTES

House Bill 2151. This bill to license chiropractors was reported by the Committee on Public Health: "no legislation necessary." However, there were seven dissenters. Actually it was a tie vote, as Senator Olander, of Northampton, did not vote, being absent because of sickness. The names of the dissenters were: Senators Montminy, of Lowell, and Skibinski, of Chicopee, and Representatives Bessette, of New Bedford, Brooks, of Lawrence, Jordan, of Revere, Lunney, of Holyoke, and Stetson, of Middleboro. The legislators who voted against the chiropractors were: Senator McCooey, of Worcester, and Representatives Bergeron, of Amherst, Cutler, of Needham, Kaplan, of Boston, Kelley, of Worcester, Vaughan, of Belmont, and Whitcomb, of West Boylston.

It may be seen from this list that the Worcester District Medical Society has done the most effective legislative work in connection with this bill.

On May 16, at the third reading of the bill in the House, the committee's report of "no legislation necessary" was accepted, and since this report was unfavorable, the bill has been referred to the Senate for concurrence.

CHARLES C. LUND, *Chairman.*

DEATH

CABOT — RICHARD C. CABOT, M.D., of 101 Brattle Street, Cambridge, died May 8. He was in his seventy-second year.

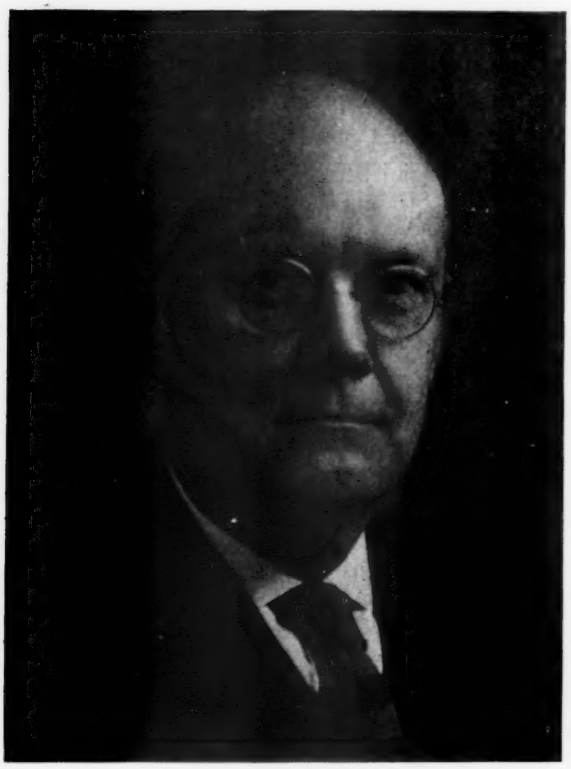
He received his degree from the Harvard Medical School in 1892. Dr. Cabot was made an assistant professor in medicine at Harvard Medical School in 1899 and in 1918 was raised to a full professor of clinical medicine. Two years later he was appointed professor of social ethics at Harvard University and held that position until his retirement in 1934.

Dr. Cabot was affiliated with the Massachusetts General Hospital and was a member of the consulting staff at the time of his death. He was also a consultant at the New England Hospital for Women and Children, the Westboro School for Boys and the Lancaster School for Girls. In 1905 he inaugurated a social-service department at the Massachusetts General Hospital; the value of this type of work was soon appreciated, and such departments were subsequently established in the majority of the large hospitals of the country.

Among his affiliations were fellowships in the Massachusetts Medical Society, the American Medical Association and membership in the Association of American Physicians.

Two brothers, Dr. Hugh Cabot and Philip Cabot, survive him.

OFFICERS OF THE MASSACHUSETTS MEDICAL SOCIETY, 1938-1939



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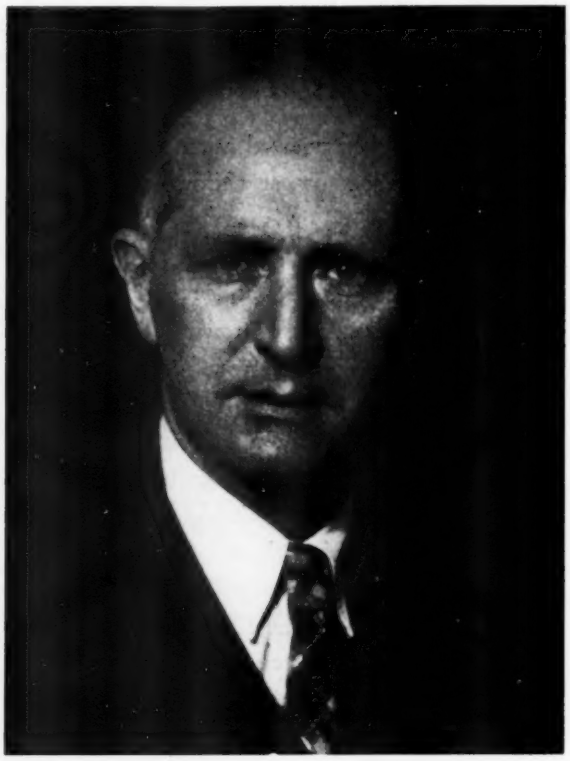

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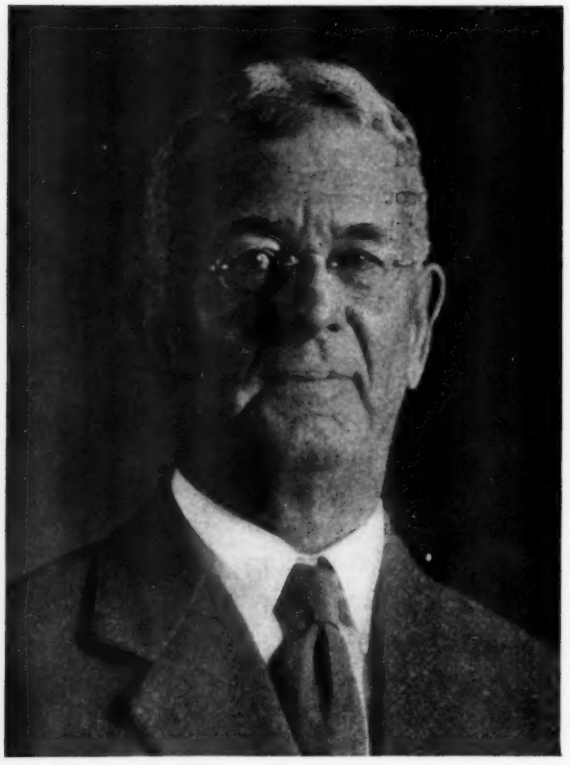

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MASSACHUSETTS MEDICAL SOCIETY

THE ONE HUNDRED AND FIFTY-EIGHTH ANNIVERSARY

Tuesday, Wednesday and Thursday, June 6, 7 and 8

Municipal Memorial Auditorium and Hotel Bancroft, Worcester

The exercises of the one hundred and fifty-eighth anniversary of the Society will be held in Worcester, on June 6, 7 and 8, at the Municipal Memorial Auditorium and the Hotel Bancroft. Members of the medical profession are cordially invited to attend.

The general arrangements for the meeting have been made by the Committee of Arrangements, consisting of Drs. Richard P. Stetson, chairman, Augustus Thorndike, Jr., Edward J. O'Brien, William T. O'Halloran and James A. Halsted. The local arrangements have been in charge of committees from the Worcester District Medical Society, with Dr. Charles A. Sparrow acting as general chairman and Dr. James C. McCann, as assistant chairman. The chairmen of the various local subcommittees are as follows:

COMBINED GENERAL MEETING AND ROUND TABLE DISCUSSIONS: Dr. James C. McCann.

SCIENTIFIC EXHIBITS: Dr. William J. Elliott.

PUBLICITY: Dr. Philip H. Cook.

TRAFFIC AND PARKING: Dr. Robert J. Northbridge.

SECTION MEETINGS AND AIDS: Dr. George R. Dunlop.

GOLF: Dr. Julius J. Tegelberg.

REFRESHMENTS: Dr. Henry L. Kirkendall.

ENTERTAINMENT: Dr. George C. Tully.

LADIES' COMMITTEE: Mrs. Charles A. Sparrow.

The registration desk will be on the stage of the Worcester Municipal Memorial Auditorium.

All section and combined meetings, as well as that of the Council, the annual meeting, commercial and scientific exhibits, and luncheons will be held in the Municipal Memorial Auditorium.

The Shattuck Lecture and the annual dinner will be held at the Hotel Bancroft.

The scientific and commercial exhibits will all be located in the main hall of the Auditorium.

A special telephone switchboard and operator will be installed in the Auditorium for the use of the fellows. The telephone number will be WORCESTER 3-1401. *Please use this number.*

A cafeteria will be maintained in the Auditorium for the use of those attending these meetings.

A golf tournament will be held at the Wachusett Golf Club on June 6 and 7.

Moving pictures of medical subjects will be shown throughout the meeting.

Windshield stickers will be sent to all fellows and cars bearing this sticker will be shown special parking consideration by the Police Department of the City of Worcester.

TUESDAY, JUNE 6

SECTION OF MEDICINE

9:45 to 11:15 o'clock

Little Theater, Worcester Memorial Auditorium

Dr. Edward P. Bagg, Holyoke, *Chairman*
Dr. Erwin C. Miller, Worcester, *Secretary.*

1. *The Importance of Hypochromic Anemia.* Dr. William P. Murphy, Boston.
2. *The Role of Oxygen in the Treatment of Pneumonia.* Dr. Alexander M. Burgess, Providence, Rhode Island (by invitation).
3. *Atypical Pneumonia of Unknown or Possibly Virus Etiology.* Dr. Michael E. Murray, Jr., Cambridge and Boston.

Committeeman-in-charge: Dr. William T. O'Halloran; aids, Drs. Roger W. Robinson and George C. Erickson.

SECTION OF DERMATOLOGY AND SYPHILOLOGY

11:30 to 1:00 o'clock

Little Theater, Worcester Memorial Auditorium

Dr. E. Lawrence Oliver, Boston, *Chairman.*
Dr. John G. Downing, Boston, *Secretary.*

1. *Chairman's Address.* Dr. E. Lawrence Oliver, Boston.
2. *Latency and Wassermann-Fastness.* Dr. Paul A. O'Leary, Mayo Clinic, Rochester, Minnesota (by invitation).
3. *The Indications and Contraindications of Roentgen Rays in Dermatology.* Dr. C. Guy Lane, Boston.

Committeeman-in-charge: Dr. James A. Halsted; aids, Drs. Gerald J. Sullivan and Gerald Shelby.

SECTION OF PEDIATRICS

2:00 to 3:30 o'clock

Little Theater, Worcester Memorial Auditorium

Dr. Elmer W. Barron, Malden, *Chairman.*
Dr. James M. Baty, Belmont and Boston, *Secretary.*

1. *Non-Specific Therapy.* Dr. Francis C. McDonald, Stoneham and Boston.
2. *Specific Therapy.* Dr. Edward C. Curnen, Boston (by invitation) and Dr. John A. V. Davies, Boston.

Discussion: Dr. Maxwell Finland, Boston.
Committeeman-in-charge: Dr. Augustus Thorndike, Jr.; aids, Drs. Smith G. Philips and Harry B. Goodspeed.

SECTION OF RADIOLOGY AND PHYSIOTHERAPY

3:45 to 5:15 o'clock

Little Theater, Worcester Memorial Auditorium

Dr. Herman A. Osgood, Boston, *Chairman.*
Dr. Edward C. Vogt, Boston, *Secretary.*

1. *Physical Therapy in the Treatment of Fractures.* Dr. John S. Coulter, professor of physical therapy, Northwestern University Medical School, Chicago, Illinois (by invitation).

2. *The Value of X-Ray in the Treatment of Infections and Inflammatory Conditions.* Dr. Arthur U. Desjardins, Mayo Clinic, Rochester, Minnesota (by invitation).

Committeeman-in-charge: Dr. Edward J. O'Brien; aids, Drs. Herman L. Matern and Charles V. King.

8:00 o'clock

Ballroom, Hotel Bancroft

THE SHATTUCK LECTURE

Epilepsy and the Cerebral Lesions of Birth and Infancy. Dr. Wilder Penfield, Montreal, director, Montreal Neurological Institute and professor of neurology and neurosurgery, McGill University Faculty of Medicine, Montreal.

Light refreshments will be served after the lecture.

Committeeman-in-charge: Dr. James A. Halsted; aid, Dr. Henry L. Kirkendall.

* * *

WEDNESDAY, JUNE 7

COMBINED MEETING

9:00 to 1:00 o'clock

Little Theater, Worcester Memorial Auditorium

Dr. James C. McCann, Worcester, *Chairman.*
Dr. James A. Halsted, Dedham, *Vice-Chairman.*

9:00 o'clock

Obstructing Lesions of the Large Bowel. Dr. Richard B. Cattell, Boston.

10:00 o'clock

Cardiac and Circulatory Failure. Dr. Arthur M. Fishberg, New York City (by invitation).

10:45 o'clock

Diagnostic Services for Equine Encephalomyelitis. Dr. Roy F. Feemster, Massachusetts Department of Public Health.

11:00 o'clock

Management of the Nervous Patient. Dr. Walter C. Alvarez, Mayo Clinic, Rochester, Minnesota (by invitation).

12:00 o'clock

Endocrines and Endocrine Therapy in Gynecology. Dr. Emil Novak, Baltimore (by invitation).

Committeeman-in-charge: Dr. James A. Halsted; aids, Drs. Arthur C. Brassau, Thomas Hunter, Edward Budnitz and Franklyn P. Bousquet.

SUPERVISING CENSORS' ANNUAL MEETING

10:00 o'clock

Green Room, Worcester Memorial Auditorium

ANNUAL MEETING OF COUNCIL

10:30 o'clock

Stage, Worcester Memorial Auditorium

This meeting will be followed by the Cotting Luncheon to councilors. Should the council meeting be prolonged, the councilors will reconvene for an adjourned meeting.

Notices of the meeting, with the order of business, will be mailed to councilors on May 29.

Committeemen-in-charge: Council Meeting, Dr. Richard P. Stetson; aid, Dr. George C. Tully; Cotting Luncheon, Dr. Edward J. O'Brien; aid, Dr. Henry L. Kirkendall.

ROUND TABLE DISCUSSIONS

Worcester Memorial Auditorium

2:00 to 3:00 o'clock

LITTLE THEATER

Craniocerebral Injuries. Chairman. Dr. Donald Munro, Boston; collaborators: Dr. John S. Hodgson, Boston, and Dr. Joseph Hahn, Springfield.

ROOM A

Heart Disease. Chairman: Dr. Samuel A. Levine, Boston; collaborators: Dr. Howard B. Sprague, Boston, and Dr. James Z. Naurison, Springfield.

ROOM B

Hyperthyroidism. Chairman: Dr. Howard M. Clute, Boston; collaborators: Dr. Lewis M. Hurxthal, Boston, and Dr. Jacob Lerman, Boston.

3:00 to 4:00 o'clock

LITTLE THEATER

Pneumonia. Chairman: Dr. Donald S. King, Boston; collaborators: Dr. Raymond H. Goodale, Worcester, and Dr. Elliott S. A. Robinson, Boston.

ROOM A

Low-Back Pain. Chairman: Dr. Louis E. Phaneuf, Boston; collaborators: Dr. John W. O'Meara, Worcester, and Dr. James B. Ayer, Boston.

ROOM B

Peripheral Vascular Disease. Chairman: Dr. Reginald H. Smithwick, Boston; collaborators: Dr. John B. Sears, Boston, and Dr. Robert R. Linton, Boston.

4:00 to 5:00 o'clock

LITTLE THEATER

Urinary Infections at Different Ages. Chairman: Dr. E. Granville Crabtree, Boston; collaborators: Dr. Roger C. Graves, Boston, and Dr. Lester M. Felton, Worcester.

ROOM A

Neonatal Injuries. Chairman: Dr. Joseph W. O'Connor, Worcester; collaborators: Dr. James S. P. Beck, Worcester, and Dr. Randolph K. Byers, Boston.

ROOM B

Adolescent Prepsychotic Conditions. Chairman: Dr. R. P. Kemble, Worcester; collaborators: Dr. George P. Reynolds, Boston, and Dr. Frederick Rosenheim, Boston.

Committeemen-in-charge: Little Theater, Dr. James A. Halsted; aids, Drs. John T. Carmody, John B. Butts and Frank B. Carr; Rooms A and B, Dr. Augustus Thorndike, Jr.; aids, Drs. Carroll W. Johnson, Edwin B. Seelye, Leroy E. Mayo, Theodore B. Massell, Edward S. Ramsdell and Clifford Guptill.

ANNUAL DINNER

7:15 o'clock

Ballroom, Hotel Bancroft

Tickets for the dinner (price $1.00) should be obtained at the Registration Desk.

Committeeman-in-charge: Dr. Edward J. O'Brien; aid, Dr. Charles A. Sparrow.

* * *

THURSDAY, JUNE 8

SECTION OF OBSTETRICS AND GYNECOLOGY

9.00 to 10.30 o'clock

Little Theater, Worcester Memorial Auditorium

Dr. Roy J. Heffernan, Boston, *Chairman.*
Dr. M. Fletcher Eades, Boston, *Vice-Chairman.*
Dr. Raymond S. Titus, Boston, *Secretary.*

1. *A Résumé of Maternal Mortality Study in the State of Massachusetts for the Year 1938.* The Secretary of the Section.
2. *Rupture of the Uterus.* Dr. Frederick J. Lynch, Boston.
3. *Obstetric Analgesia.* Dr. Benjamin F. Cornwall, Salem.

Committeeman-in-charge: Dr. William T. O'Halloran; aids, Drs. Donald K. McClusky and Herbert E. Hedberg.

SECTION OF SURGERY

10:30 to 12:00 o'clock

Little Theater, Worcester Memorial Auditorium

Dr. Benjamin H. Alton, Worcester, *Chairman.*
Dr. Reginald H. Smithwick, Boston, *Secretary.*

SURGERY OF THE STOMACH AND DUODENUM

1. *Medical Aspects.* Dr. Chester M. Jones, Boston.
2. *Gastroscopic Examination.* Dr. Edward B. Benedict, Boston.
3. *X-Ray Examination.* Dr. William J. Elliott, Worcester.
4. *Surgery of the Stomach.* Dr. Arthur W. Allen, Boston.
5. *Surgery of the Duodenum.* Dr. Frank H. Lahey, Boston.

Discussion by: Dr. Philemon E. Truesdale, Fall River, and Dr. James C. McCann, Worcester.

Committeeman-in-charge: Dr. Richard P. Stetson; aids, Drs. John W. McKoan, Jr., and Earl E. Fipphen.

ANNUAL MEETING

12:00 o'clock

Little Theater, Worcester Memorial Auditorium

Business of the Annual Meeting.

Address by the President.

Annual Discourse: ***The Massachusetts Medical Society and Socialized Medicine.*** Dr. Elliott P. Joslin, Boston.

Committeeman-in-charge: Dr. Augustus Thorndike, Jr.; aid, Dr. George C. Tully.

At the close of the Annual Discourse, luncheon will be served to those who have obtained tickets at the Registration Desk.

Committeeman-in-charge: Dr. William T. O'Halloran; aid, Dr. Carroll W. Johnson.

LADIES' PROGRAM

TUESDAY — JUNE 6

9:00–12:00	Registration, Worcester Memorial Auditorium.
12:00	Bus leaves main entrance of Auditorium for: 1. Luncheon at the Worcester Country Club. 2. The gardens of Mrs. Emory and Mrs. Gage in Shrewsbury.
6:15	Dinner at the Worcester Club, 1 Oak Street.
8:00	Shattuck Lecture, Hotel Bancroft, by Dr. Wilder Penfield, Montreal.

WEDNESDAY — JUNE 7

9:00–12:00	Registration, Worcester Memorial Auditorium.
12:00	Bus will leave the Auditorium to take visitors to luncheon at the "Barn," Harvard, Massachusetts, and a tour of three museums: 1. "Fruitlands" (home of the Alcott family). 2. Shaker Exhibit. 3. American Indian Museum. Tea will be served at the Museum Tea Room.

8:15 Ladies are invited to hear the speakers after the annual dinner of the Massachusetts Medical Society at the Hotel Bancroft. The gallery will be reserved for their use.

There will be no charge for any of the events. The women visitors will be the guests of the Massachusetts Medical Society and the Worcester District Medical Society.

MOVING PICTURES

Main Floor, Worcester Memorial Auditorium

Warren Sturgis, *Director.*

TUESDAY — JUNE 6

10:00 *The Prevention and Treatment of Eclampsia.*
10:45 *Complications of the Second Stage of Labor.*
11:00 *The Treatment of Asphyxia Neonatorum.*
11:30 *Intracranial Injuries of the Newborn.*
12:00 *The Valves of the Heart in Action.*
12:15 *The Heart Mechanism in Health and Disease.*
12:30 *Ectopic Heart.*
12:45 *Thrombi and Emboli.*
1:00 *The Technic of Blood Transfusion.*
1:30 *The Anemias.*
2:00 *Intestinal Peristalsis.*
2:15 *The Action of Drugs on Intestinal Motility.*
2:30 *Bronchoscopy and Esophagoscopy on the Cadaver and on the Living.*
2:45 *Obstructive Laryngitis.*
3:00 *Oxygen Administration by the Oropharyngeal Catheter.*
3:15 *Myasthenia Gravis.*
3:30 *Teaching Diabetics.*
3:45 *Development of the Fertilized Rabbit's Ovum.*
4:00 *The Diagnosis and Treatment of Syphilis.*
4:30 *A New Day (Pneumonia Serum Treatment).*

WEDNESDAY — JUNE 7

10:00 *Child Guidance Work.*
10:15 *Reconstruction of Crippled Hands.*
11:00 *Pneumonectomy.*
11:15 *Empyema.*
11:30 *Diagnostic Procedures in Tuberculosis.*
11:45 *On the Firing Line (Tuberculosis Public-Health Work).*
12:00 *Hernia: Clinical and operative aspects.*
12:30 *The Mechanics of Appendicitis.*
12:45 *Appendectomy for Acute Gangrenous Appendicitis.*
1:00 *Treatment of Burns.*
1:30 *First Aid for Safe Transportation of Fractures of the Long Bones.*
1:45 *Treatment of Fractures in Children.*
2:00 *Bone-Plating of Fractured Radius.*
2:15 *Protruded Intervertebral Disk.*
2:30 *Aftercare of Poliomyelitis.*
3:00 *Lower Limb Amputation and Aftercare of the Patient.*
3:30 *Human Sterility.*
4:15 *The Aschheim-Zondek Pregnancy Test.*
4:30 *Athetosis.*

This program is subject to change without notice. The times given are approximate.

SCIENTIFIC EXHIBITS

Main Auditorium, Worcester Memorial Auditorium

	Booth
Cutaneous Manifestations of Syphilis. American Medical Association	A & B
Medical Education. American Medical Association	C
Patent Medicines and Quackery. American Medical Association	D & E
A. M. A. Council on Pharmacy and Chemistry. American Medical Association	F
A. M. A. Council on Physical Therapy. American Medical Association	G
A. M. A. Council on Foods. American Medical Association	H
Transurethral Resection of the Prostate and Roentgen Visualization of the Prostate and Urethra. Drs. Fletcher H. Colby and Howard I. Suby; Urological Service, Massachusetts General Hospital, Boston	I
Cancer. Pondville Hospital, Massachusetts Department of Public Health	J
The Background of Scientific Milk Production. Medical milk commissions of Boston, Worcester and Springfield	K
Fractures. Dr. Gordon M. Morrison, Boston	L
Cancer Teaching Exhibit. Boston Dispensary, New England Medical Center	M & N
Division of Child Hygiene. Massachusetts Department of Public Health	O
Services for Crippled Children. Massachusetts Department of Public Health	P
Dermatitis Artefacta and Dermatitis Factitia. Dr. John G. Downing, Boston	Q
Observations on the Effect of Insulin in Nondiabetic Malnutrition. Dr. Harry Blotner, Medical Clinic of the Peter Bent Brigham Hospital, Boston	R
Roentgenological and Pathological Studies of Pulmonary Conditions. Rutland State Sanatorium, Rutland	S
Research in Mental Disease: Cardiotachometer and encephalogram. Worcester State Hospital, Massachusetts Department of Mental Health	T
Urinary Pathology. Drs. Lester M. Felton, Walter D. Bieberbach, Bancroft C. Wheeler and Francis J. Steele, Worcester	U
Lumbosacral Pathologic Lesions as Causes of Sciatica. Dr. Charles E. Ayers, Worcester	V
Blind Nailing of Fractured Hips. Drs. John W. O'Meara, James A. Givan and Charles V. King, Worcester	W
Bilateral Fractures of the Femur. Orthopedic Service, Worcester City Hospital	X

COMMERCIAL EXHIBITS

Main Auditorium, Worcester Memorial Auditorium

Booth No.

1. Ernst Bischoff Company, Incorporated, Ivoryton, Connecticut.

In our exhibit we shall feature: Activin, the first American-produced shockless foreign protein for nonspecific therapy; Alpha-Lobelin, a direct stimulant to the respiratory tract and the resuscitant indicated in all forms of respiratory failure or depression; Diatussin, the original drop-dose cough remedy with a thirty-five-year record of efficacy; Silvogon, an absolutely stainless silver antiseptic, an effective gonocide; Styptysate, a vegetable hemostatic for the control of all seeping hemorrhages; Viscysate, a dialysate of viscum album which lowers blood pressure and relieves the accompanying symptoms.

2. Philip Morris and Company, Limited, Incorporated, New York City.

Philip Morris and Company will demonstrate the method by which it was found that Philip Morris Cigarettes, in which diethylene glycol is used as the hygroscopic agent, are less irritating than other cigarettes. Its representative will be happy to discuss researches on this subject, and problems on the physiologic effects of smoking.

3. Hynson, Westcott and Dunning, Incorporated, Baltimore, Maryland.

Hynson, Westcott and Dunning will have an exhibit featuring Mercurochrome and various pharmaceutical specialties of their manufacture. There will also be a display of some of the diagnostic apparatus and ampule solutions which have been developed in cooperation with physicians. As usual, competent representatives of the company will be in attendance to demonstrate the products and to answer questions. Literature and samples will be available to physicians who are not already familiar with products exhibited or who wish to obtain a trial supply.

4. Moore and Company, Incorporated, Worcester, Massachusetts.

Moore and Company will display pharmaceutical preparations and specialties all of which are guaranteed to be true to label and conform to the requirements of the federal and state laws pertaining to the standards and purity of drugs.

5. Campbell X-Ray Corporation, Boston.

Campbell X-Ray Corporation will exhibit new methods of shock proofing x-ray apparatus.

6. The E. L. Patch Company, Stoneham, Massachusetts.

The Patch Company representatives will be on hand throughout the meeting to greet physicians and to be of service in any way. The Patch Company exhibit will include Patch's Cod-Liver Oil, as well as the other ethical medicinal specialties made in the Patch Laboratory.

7. Kanef Drug Company, Worcester, Massachusetts.

The Kanef Drug Company will exhibit pharmaceutical and biological products manufactured by Abbott Laboratories; Lederle Laboratories, Incorporated; Sharp and Dohme; and Winthrop Chemical Company, Incorporated.

8-9. The P. L. Rider Company, Worcester, Massachusetts.

The P. L. Rider Company will display a complete line of physicians' office equipment, surgical instruments, diagnostic instruments and specialties, as well as trusses, sacroiliac and abdominal supports, stockings, suction and pressure pumps, and electrical centrifuges.

10. Eli Lilly and Company, Indianapolis, Indiana.

Eli Lilly and Company feature an eight-foot exhibit stressing the importance of liver extract in the treatment of pernicious anemia, Merthiolate (sodium ethyl mercuri thiosalicylate, Lilly) in the surgical and germicidal fields, Sodium Amytal (sodium iso-amyl ethyl barbiturate, Lilly) in the field of hypnotics, and Iletin (insulin, Lilly) in the management of diabetes mellitus. This is the first appearance of the Lilly Research Laboratories at the meetings of the Massachusetts Medical Society; the exhibit unit has been specially designed for state medical meetings.

11. Jones Metabolism Equipment Company, New York City.

The Jones Metabolism Equipment Company presents the most modern metabolism apparatus on the market. The Jones Motor-Basal eliminates corrections for barometric pressure and room temperature, and eliminates calculations. It is so simple that anyone can learn to run it in a short time and yet is accurate enough to meet the most exacting requirements of research laboratories. An exclusive geometric device checks the accuracy of each test, thus eliminating the possible error caused by the human element. The Jones Motor-Basal is guaranteed to perform with an accuracy greater than 99 per cent for the lifetime of the purchaser.

12. Davies, Rose and Company, Limited, Boston.

Members of the Massachusetts Medical Society are so well acquainted with the products of the laboratory of Davies, Rose and Company that no further explanation of their merits is really necessary. However, the company trusts that you will visit its booth and give its representatives the honor of greeting you. Messrs. Mansfield and Fleming will be in attendance.

13. Baby's Dy-Dee Service, Incorporated, Brookline, Massachusetts.

This exhibit explains in detail the specialized equipment and methods used in sterilizing and supplying diapers to homes in Greater Boston. Starting its sixth year, this service has relieved hundreds of mothers and nurses of the daily drudgery of washing diapers and baby clothes, at the same time protecting baby's health by scientific methods impossible at home. An economical service, devoted exclusively to the baby.

Represented by Mary Eustis Sturgis and Faye Reynolds Rand.

14-15. John Wyeth and Brother, Incorporated, Philadelphia.

John Wyeth and Brother will display the following pharmaceutical specialties from their list of products: Kaomagma, an intestinal adsorbent particularly useful in checking diarrhea; Amphojel, Wyeth's aluminum hydroxide for the treatment of gastric hyperacidity and peptic ulcers; Silver Picrate Wyeth, used in the treatment of trichomonas vaginitis; Bewon Elixir, a palatable form of crystalline vitamin B_1; Mucara, a processed form of karaya gum used for

habitual constipation; and other products of general interest to practitioners and specialists.

16. M and R Dietetic Laboratories, Incorporated, Columbus, Ohio.

M and R Dietetic Laboratories will display Similac, a completely modified milk for infants deprived of breast feeding. Representatives will gladly explain its merit and suggested application.

17. E. R. Squibb and Sons, New York City.

Physicians are cordially invited to visit the Squibb Exhibit. The complete line of Squibb vitamin, glandular, arsenical and biological products and specialties, as well as a number of interesting new items will be featured. Well-informed Squibb representatives will be on hand to welcome you and to furnish any information desired on the products displayed.

18. Lea and Febiger, Philadelphia.

Among the new books which Lea and Febiger will exhibit, in charge of Mr. Paul Loveday, are Stimson's *Fractures and Dislocations;* Haden's *Hematology;* Miller's *Applied Anatomy;* Cowan's *Refraction of the Eye;* Schlanser's *Practical Otology, Rhinology and Laryngology;* Thorndike's *Athletic Injuries* and Brenner's *Pediatric Surgery.* New editions will be shown of MacKee's *X-Ray Therapy;* Pohle's *Clinical Roentgen Therapy;* Pohle's *Theoretical Principles of Roentgen Therapy;* Crotti's *Diseases of the Thyroid, Parathyroids and Thymus;* Kovacs's *Electrotherapy and Light Therapy;* Ballenger's *Nose, Throat and Ear;* Ivy and Curtis's *Fractures of the Jaw;* Brown's *The Surgery of Oral and Facial Diseases and Malformations* and Kanaval's *Infections of the Hand.*

19. H. J. Heinz Company, Pittsburgh.

Heinz Junior Foods, a new variety for older babies, are on display. The Heinz representative is ready to assist you to inspect this new product, as well as the Heinz Strained Foods, also on display. Register at the Heinz booth for helpful literature.

20. Mellin's Food Company, Boston.

Opportunity will be offered for a discussion of the application of Mellin's Food in the feeding of infants whose individual condition sets them apart from so-called normal babies, and whose diet needs to be adjusted in a manner calculated to correct their digestive disturbance. Mellin's Food is worthy of attention for it has occupied an outstanding position in the field of pediatrics ever since the beginning of the study of the art and science of infant feeding.

21. The Borden Company, New York City.

Full information on Biolac, the new liquid modified milk for infants, will be available at the Borden booth. Also exhibited will be other Borden products for infant feeding, notably Klim, Dryco, Beta Lactose, Merrell-Soule products and Borden's Silver Cow Evaporated Milk.

22. Crosbie–Macdonald, Boston.

We represent the United States Fidelity and Guaranty Company, writing physician's liability insurance for members of the Massachusetts Medical Society. Either George H. Crosbie, Edward J. O'Neil, Jr., or Arthur H. Crosbie will be on hand at all times to discuss any questions pertaining to insuran e and to quote rates for your specialty. We are notaries public, and should be glad to sign and file your narcotic drug blanks.

23-24. Pet Milk Company, Saint Louis, Missouri.

An actual working model of a milk-condensing plant in miniature will be exhibited by the Pet Milk Company. This exhibit offers an opportunity to obtain information about the production of Irradiated Pet Milk and its uses in infant feeding and general dietary practice. Miniature Pet Milk cans will be given to each physician who visits the Pet Milk booths. Our representatives, Mr. D. O. Tracy and Mr. G. F. Whalen, will be in charge of the exhibit.

25. Tailby–Nason Company, Boston.

Tailby–Nason Company has reserved space for the exhibit of Nason's Palatable Cod Liver Oil, made in the company's own plants in the Lofoten Islands of Norway, romantic Land of the Midnight Sun.

More and more leading physicians are relying on good cod-liver oil in all cases requiring vitamins A and D. Nason's Oil is prescribed and recommended by leading pediatricians from the Atlantic to the Pacific for its high vitamin potency and unusual palatability.

26. S.M.A. Corporation, Cleveland, Ohio.

This interesting new display represents the selection of infant feeding and vitamin products of the S.M.A. Corporation. Physicians who visit this exhibit may obtain complete information, as well as samples, of S.M.A. powder and special milk preparations — Protein S.M.A. (Acidulated), Alerdex and Hypo-Allergic Milk.

27. The Arlington Chemical Company, Yonkers, New York.

Again The Arlington Chemical Company will feature its biological and pharmaceutical products. They are offering a $9.75 diagnostic protein outfit, consisting of eighty of the commonest causative factors in allergic conditions; also a full line of food and fungus proteins and pollen extracts for diagnosis and desensitization. The representative in charge of the exhibit will be glad to discuss any allergic problem.

28. Smith, Kline and French Laboratories, Philadelphia.

Smith, Kline and French Laboratories, believing that many physicians dislike efforts to make them register, have arranged their booth for self-service. Information about Benzedrine Inhaler, Benzedrine Sulfate Tablets, Benzedrine Solution, Pentnucleotide, Feosol Tablets, Feosol Elixir, Oxo-ate "B" Tablets and Eskay's Neuro Phosphates may be obtained from the convenient literature dispenser. If additional information is desired, the representative will be glad to answer any questions.

29. J. Sklar Manufacturing Company, Brooklyn, New York.

The Sklar Manufacturing Company exhibit will feature new suction and pressure apparatus, including the improved Tompkins portable rotary compressor, the de luxe Tompkins, the new Imperatori apparatus

for ear, nose and throat work, the Ralks Ideal Unit and the Moorhead unit for office and clinic, and the new, improved heavy-duty hospital model of the Bellevue suction and pressure unit. The Sklar Company will also exhibit its extensive line of American made stainless-steel surgical instruments, as well as special apparatus, such as the Davidson pneumothorax apparatus, the Soresi blood transfuser, and so forth.

30. The Macmillan Company, New York City.

You are cordially invited to visit the exhibit of the Macmillan Company. On display at this booth you will find two outstanding new medical books, which the Macmillan representative will be glad to discuss with you. One is *The Experimental and Clinical Use of Sulphanilamide, Sulphapyridine and Allied Compounds,* by Perrin H. Long, M.D., and Eleanor A. Bliss, Sc.D. The timeliness of the subject and the unquestionable authority of the authors should give this book an immediate claim to your attention. The other is *Otolaryngology in General Practice,* by Lyman Richards, M.D. An examination of the books with their unique arrangement and fine illustrations will impress you with their practical helpfulness.

Also on display will be the new fifth edition of Dr. Hans Zinsser's *Resistance to Infectious Diseases,* now significantly entitled *Immunity: Principles and application in medicine and public health* and written in collaboration with John F. Enders, Ph.D., and LeRoy D. Fothergill, M.D.

31. J. B. Lippincott Company, Philadelphia.

Among the newer Lippincott publications on display are the phenomenally successful Thorek's *Modern Surgical Technic* and Kracke's *Diseases of the Blood and Atlas of Hematology,* from which illustrations are being displayed at the World's Fair Medical Exhibit. Other important new works include: Rigler's *Outline of Roentgen Diagnosis,* Barborka's *Treatment by Diet* and Imperatori's *Diseases of the Nose and Throat.*

32. The Alkalol Company, Taunton, Massachusetts.

The Alkalol Company will exhibit two preparations: Alkalol, a carefully balanced solution of alkaline and saline salts and essential oils, which is especially prepared for use on mucous membranes or on inflamed or irritated tissues, and Irrigol, a powder which makes an alkaline, saline, non-toxic solution and is valuable for vaginal douches, rectal enemas or colonic irrigations.

33. Frederick Stearns and Company, Detroit, Michigan.

Frederick Stearns and Company will exhibit their Neo-Synephrin products, Mucilose, Trimax, Apple Powder and Gastric Mucin and will feature their newly developed product — Solution Zinc-Insulin Crystals, Stearns. Mr. J. P. Burfeind will be in charge.

34. Parke, Davis and Company, Detroit, Michigan.

Members of the staff of Parke, Davis and Company will be at your service to tell you about some of their research staff's numerous scientific accomplishments. Mapharsen, Adrenalin, Pitocin, Pitressin, Theelin, Theelol and biological products will be a part of this attractive exhibit.

35. Petrolagar Laboratories, Incorporated, Chicago.

Petrolagar Laboratories offer, in addition to samples of the five types of Petrolagar, an interesting selection of descriptive literature and anatomical charts. Ask the Petrolagar representative, Mr. E. M. Tarplin or Mr. G. E. Schneider, to show you the new *Habit Time* booklet. It's a welcome aid for teaching bowel regularity to your patients.

36. General Electric X-Ray Corporation, Boston.

The General Electric X-Ray Corporation will exhibit a miniature model darkroom, an electrocardiograph and a portable x-ray machine.

37. White Laboratories, Incorporated, Newark, New Jersey.

The White Laboratories will present information covering the entire field of cod-liver oil concentration, together with clinical data and evidence concerning the therapeutic efficacy of its Cod Liver Oil Concentrate, in liquid, tablet and capsule form. Informed representatives and descriptive literature, reprints and excerpts will further demonstrate cod-liver oil efficacy, and point out the contributions of White Laboratories in the vitamin A and vitamin D field.

White Laboratories is the world's largest manufacturer of cod-liver oil concentrates and is one of the largest users of cod-liver oil for pharmaceutical purposes in the world. All physicians are cordially invited to visit the booth.

38. Westinghouse X-Ray Company, Incorporated, Long Island City, New York.

The Westinghouse X-Ray Company will exhibit several new diagnostic x-ray items that will be of interest to all who are doing x-ray work. There will be shown for the first time an automatic head clamp which was originally developed at Temple University and has proved to be of great value to radiologists and therapists.

39. American Hospital Supply Corporation, Chicago.

The new simple Baxter blood-transfusion set will be shown. See the Baxter Vacoliter, the same intravenous solution used exclusively by many teaching institutions and by over half of all American hospitals. Investigate Coli-Bactragen, it *does* prevent peritonitis. Let us demonstrate the Oxygenaire and the Tomac oxygen insufflator. Look over the automatic apparatus for continuous Wangensteen suction. *Don't miss this booth.*

40. Lee De Forest Laboratories, represented by the New England X-Ray Corporation, Boston.

Lee De Forest Laboratories will demonstrate the ultimate in radiotherapy and radiosurgery, designed and built by radio scientists and engineers. Mr. George Laben will be in charge of the exhibit.

41. Mead, Johnson and Company, Evansville, Indiana.

Three new Mead products are on display at Mead Johnson and Company's booth: Mead's Thiamin Chloride Tablets; Mead's Cevitamic Acid Tablets; Mead's Nicotinic Acid Tablets. Olac for feeding premature babies is also shown, as well as the complete line of Mead's infant-diet materials.

42. Lederle Laboratories, Incorporated, New York City.

Lederle Laboratories will exhibit a full line of biologicals and pharmaceutical specialties featuring the new drug sulfapyridine.

43. Winthrop Chemical Company, Incorporated, New York City.

The Winthrop Chemical Company extends a cordial invitation to every member of the Massachusetts Medical Society to visit its booth where representatives will gladly discuss the latest preparations made available by this firm. You will receive valuable booklets dealing with anesthetics, analgesics, antirachitics, antispasmodics, antisyphilitics, diagnostics, diuretics, hypnotics, sedatives and vasodilators.

44. Surgeons' and Physicians' Supply Company, Boston.

The Surgeons' and Physicians' Supply Company will exhibit the new Comprex short-wave apparatus, with cable, and other new and interesting items, both in the line of instruments and in that of general supplies.

45. Riedel-de Haen, Incorporated, New York City.

Riedel-de Haen, the pioneers in bile-acid research, will feature the bile-acid products, Decholin, Decholin Sodium and Degalol. The hypnotics Pernoston and Pernoston Sodium will also be shown, as well as Sigmodal, the soporific for rectal administration in obstetrics and surgery.

46. Gerber Products Company, Fremont, Michigan.

The new Gerber Cereal Food, dry, pre-cooked, will be shown to you at the Gerber booth. Samples and professional literature about this cereal product, as well as the other Gerber baby foods, will be available to you.

47. Picker X-Ray Corporation, Boston.

To the roentgen profession the name of Picker–Waite has always been synonymous with progress. The first successful shockproof x-ray apparatus ever built was of Waite invention. Production facilities plus the combined resources of the Picker–Waite organization, with an electrical and mechanical engineering background of over fifty-nine years, have made it possible to produce x-ray equipment at a modest cost.

One of the most outstanding pieces of Picker–Waite equipment is the Picker–Waite Century (100 milliamperes — 100 kilovolts), which has been presented to the medical profession as a truly modern diagnostic x-ray apparatus — introducing for the first time an entirely new principle of flexibility in radiographic-fluoroscopic x-ray design and development. Point for point and dollar for dollar the Picker–Waite Century is easily the most outstanding value in diagnostic x-ray equipment offered to a discriminating clientele. We are proud to exhibit this unit with many new and outstanding smaller accessory devices at the meeting of the Massachusetts Medical Society. Bulletins and descriptive literature may be obtained at our booth.

48. Horlick's Malted Milk Corporation, Racine, Wisconsin.

Nourishing, digestible, appetizing — these are the three outstanding qualities for which Horlick's Malted Milk is famous, whether in powder or tablet form. Visit the exhibit. You will be interested in the many uses from infant feeding to old age; note especially the convenience of the tablets in ulcer diets.

49-50. Brewer and Company, Incorporated, Worcester, Massachusetts.

Thesodate (Brewer) will be featured at the booth of Brewer and Company. Since the introduction of this product one year ago, it has become the xanthine of choice with many physicians. For information and a liberal supply for clinical trial, register at the Brewer booth.

51. G. D. Searle and Company, Chicago.

Products of research, originated in the Searle laboratories, will be the feature of the exhibit of G. D. Searle and Company. Particularly interesting are the recent work on the bile acids and information on the use of Aminophyllin-Searle, as well as the display of Bismuth Sodium Tartrate-Searle.

52. Hanovia Chemical and Manufacturing Company, Newark, New Jersey.

While at the convention do not fail to call at the Hanovia booth. There will be a complete line of the most modern equipment, consisting of ultraviolet quartz lamps with special treatment control, short-wave heat generators, Sollux radiant-heat lamps and Safe-T-Aire ultraviolet lamps for air sanitation. Courteous and competent representatives will be on hand to welcome you.

53. Bilhuber–Knoll Corporation, Jersey City, New Jersey.

New reports on the fine medicinals of the Bilhuber–Knoll Corporation are always of interest. Its products include Dilaudid Hydrochloride, an opiate for pain and cough; the well-tolerated purine salts, Theocalcin and Phyllicin, so useful in the treatment of heart diseases; the cardiorespiratory restorative, Metrazol, of value as an antidote to depressant drugs, for denarcotization after anesthesia, and in the emergencies of heart failure; and the non-barbiturate sedative and hypnotic, Bromural.

Well-informed representatives will be in attendance to discuss the new reports with interested physicians.

54. The Liebel-Flarsheim Company, Cincinnati, Ohio.

The Liebel-Flarsheim Company will exhibit a complete line of the well-known L-F short-wave generators, as well as the famous Bovie electrosurgical units. In addition, other new and useful physiotherapy apparatus will be shown. A cordial invitation is extended to visit the Liebel-Flarsheim booth in order to inspect this new apparatus and have it demonstrated to you.

55. Sandoz Chemical Works, Incorporated, New York City.

The Sandoz Chemical Works will feature the following preparations: Gynergen (ergotamine tartrate) for the dramatic relief of migraine; Calglucon (calcium gluconate) granules, effervescent tablets and chocolate-flavored tablets; Neo-Calglucon, a new highly water-soluble derivative of calcium gluconate, for parenteral calcium therapy; the three reliable belladonna products, Bellafoline, Belladenal and Bellergal; Calcibronat, an organic combination of calcium and bromine; Quinine-Calcium, Sandoz; Scillaren and Scillaren-B, the cardioactive principle of squill (*Urginea maritima*); Digilanid, the crystallized initial glucosides of *Digitalis lanata;* Basergin, a stable preparation of pure ergonovine; Neo-Gynergen, a combination of ergotamine and ergonovine for obstetric and gynecological use.

56-57. E. F. Mahady Company, Boston.

The exhibit of the E. F. Mahady Company includes the latest developments in physical therapy equipment manufactured by the Burdick Corporation, as well as Cutter's intravenous solutions in Saftiflask dispensers, new surgical instruments and medical books.

58. The C. B. Fleet Company, Incorporated, Lynchburg, Virginia.

Phospho-Soda (Fleet) is a highly concentrated and purified, aqueous solution of sodium phosphates. It is non-toxic, rapid and mild in action without irritation of the gastric or intestinal mucosa. It is indicated for hepatic dysfunction, and for its thorough eliminating and cleansing action on the upper and lower intestines. The C. B. Fleet Company thanks the medical profession for the broader usage now being given to Phospho-Soda (Fleet). Its prompt, acceptable and controllable action makes it an exceptionally efficient laxative saline and cholagogue.

Mr. R. S. Carman will again extend our courtesies.

59. The Medical Protective Company, Wheaton, Illinois.

The most exacting requirements of adequate liability protection are those of the professional liability field. The Medical Protective Company, specialists in providing protection for professional men, invites you to confer, at its exhibit, with the representative there. He is thoroughly trained in professional liability underwriting.

60. The Coca-Cola Company, Atlanta, Georgia.

The Coca-Cola Company in serving complimentary Coca-Cola at its booth hopes that "The Pause That Refreshes" will be enjoyed by guests and visitors of the meeting.

61. Kalak Water Company of New York, Incorporated, New York City.

Visit the exhibit of the Kalak Water Company and test for yourself the palatability of this delicious, sparkling, neutralizing agent when served properly cooled. Kalak Water has been used by the profession for almost a quarter of a century to re-establish and maintain the alkali reserve and also to supply fluid to patients without producing overloading of the circulation. Because Kalak presents a proper balance of buffer salts, such as the bicarbonates of sodium, calcium and magnesium, it is ideally suited for use in buffering the untoward effects of sulfanilamide or sulfapyridine. Ask the Kalak representative for literature covering the buffering value of Kalak. It is important to remember that Kalak Water is not a laxative.

62. Jetter and Scheerer Products, Incorporated, New York City.

The world-famous line of Jetter and Scheerer surgical instruments will be exhibited again this year. A large variety of both rustless-steel and chrome-plated instruments will be displayed, including many unusual and special items. The Jetter and Scheerer line has been held in highest esteem by surgeons since 1867 and is still considered to be the standard for which all others aim. You are cordially invited to visit our booth.

63. Sharp and Dohme, Incorporated, Philadelphia.

Sharp and Dohme will feature their well-known Propadrine Hydrochloride products. There will also be on display a group of pharmaceutical specialties and biologicals prepared by this house. Capable, well-informed representatives will be on hand to welcome physicians and furnish information on Sharp and Dohme products.

64. Abbott Laboratories, North Chicago, Illinois.

A hearty welcome awaits you in the Abbott booth where a comprehensive assortment of Abbott specialties is on display. Abbott-trained representatives will be glad to answer questions and to discuss their newer products with you.

BOSTON UNIVERSITY SCHOOL OF MEDICINE ALUMNI ASSOCIATION

A meeting and luncheon of the Boston University School of Medicine Alumni Association will be held at 12:30 p. m., Tuesday, June 6, at the Hotel Bancroft. The charge for the luncheon will be $1.00.

HARVARD MEDICAL ALUMNI ASSOCIATION

The annual meeting and luncheon of the Harvard Medical Alumni Association will be held at 12:30 p. m., Tuesday, June 6, at the Hotel Bancroft. The charge for the luncheon will be $1.00.

MASSACHUSETTS MEDICO-LEGAL SOCIETY

A meeting of the Massachusetts Medico-Legal Society will be held at 2:00 p. m., Wednesday, June 7, in the Female Chorus Room, Second Floor, Worcester Memorial Auditorium.

TUFTS COLLEGE MEDICAL SCHOOL ALUMNI

The annual luncheon of the Tufts College Medical School Alumni Association will be held at 12:00 noon, Tuesday, June 6, on the Stage of the Worcester Memorial Auditorium. Members and guests are invited.

SECTION OF OBSTETRICS AND GYNECOLOGY*

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

BLEEDING IN THE PUERPERIUM

Mrs. A. K., a thirty-two-year-old gravida I, was delivered by low forceps on March 30, 1934.

The family history was non-contributory. The patient had had pneumonia as a child. She had never undergone an operation. Catamenia began at thirteen, were regular with a twenty-eight-day cycle, and lasted three to five days without pain.

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

Her last period was August 6, 1933, making her due for delivery early in May.

The patient was seen routinely throughout her pregnancy. Her blood pressure never went above 128 systolic, 66 diastolic, and her urine on only one occasion showed a very slight trace of albumin.

Ten days after delivery she began to flow very freely. When seen about half an hour after the flowing had started, the pulse was 100, and the blood pressure 100 systolic, 60 diastolic. She looked poorly, but as she was not flowing when seen, the uterus was not invaded. Intravenous glucose was given, and a transfusion postponed. It was believed that the bleeding was due to a piece of placenta which had not been extruded at the time of labor. The next morning her temperature was 101.6°F., having been normal before the bleeding episode. She was passing small pieces of what appeared to be retained placenta. The temperature was normal the following day.

On the next day, April 12, the patient again began to bleed very freely. She was given posterior pituitary extract, ergot and morphine. The blood pressure was 118 systolic, 70 diastolic, and the pulse 100. Under nitrous oxide, oxygen and ether anesthesia a large piece of placenta was found extruding from the cervix. This was removed manually, as were other pieces which were adherent. An alcohol pack was left in the uterus, with the hope that if anything had been left behind it would come away on the pack when removed. The pack was removed the following morning, and because of her anemia, — the hemoglobin was 40 per cent, and the red-blood-cell count 1,280,000, — transfusion was performed, a professional donor being used. Subsequent convalescence was uneventful. On April 14 the hemoglobin was 35 per cent, and the red-blood-cell count 1,090,000, and on April 17, 60 per cent and 2,360,000. There was no further rise in temperature. and no more bleeding occurred. She was discharged on April 20.

Comment. The treatment of this particular case is open to just criticism. Even though the initial bleeding had ceased when the patient was seen, it was evident that a great deal of blood had been lost. She should have been transfused at that time, and the pieces of tissue removed. This would have prevented the second hemorrhage which resulted in a marked anemia. It must be borne in mind that any patient who bleeds nine or ten days following the birth of a baby does so because of some abnormality inside the uterus. Whether one procrastinates and does not invade the uterus should depend entirely on the amount of bleeding. If the bleeding is slight, oxytocics may be used successfully; if the bleeding is profuse and no pieces of tissue are observed in the blood which is passed, one must infer that foreign material still remains.

Hemorrhage is the only indication for entering the uterus after delivery, and while one should be conservative about it, one must appreciate that any of these cases may continue to bleed until the uterus is invaded and freed of its foreign material. It is possible in such cases that an accreta is the underlying cause; if so, hysterectomy is the treatment of choice. The majority of delayed hemorrhages, however, are caused by pieces of partially adherent placenta, and these can almost invariably be removed with the fingers or with ovum forceps. Curettage, if employed, should be most delicate, and sharp instrumentation should always be avoided. Transfusion is an extremely important adjunct to intrauterine instrumentation.

MISCELLANY

THE PROFESSIONAL CLUB

Physicians, public-health workers, medical scientists and other professionals visiting the New York World's Fair 1939 will find reserved for their exclusive use the Professional Club, where members have a place to meet their colleagues in quiet, congenial surroundings.

Unique to this or any other world's fair, the club occupies an area of 5000 square feet on the main floor of the Medical and Public Health Building, which is located on the Theme Plaza, its main entrance being directly opposite the Helicline leading around the Perisphere to the Trylon, where much of importance takes place daily.

The visitor will find awaiting him a comfortable lounge, attractively decorated and furnished, a bar and a snack bar, checking facilities, rest rooms, stenographic service, telephones and other conveniences of a private club.

Membership in the club is limited to accredited members of the medical and public-health and allied professions and to representatives of exhibit sponsors. Professional members pay no dues, but there is a small certification charge to cover the cost of validating credentials. Among the organizations whose membership cards entitle their owners to admittance to and use of the club are: American Dental Association; American Dental Hygienists Association; American Dietetic Association; American Hospital Association; American Medical Association; American Medical Library Association; American Nurses Association; American Pharmaceutical Association; American Public Health Association; American Veterinary Medical Association; Association of Women in Public Health; Catholic Hospital Association; National League of Nursing Education; National Organization for Public Health Nursing; Pan-American Medical Association, Incorporated; United States Department of Agriculture, Bureau of Animal Industry; and the United States Public Health Service.

Products of manufacturers sponsoring scientific and educational exhibits in the Medical and Public Health Building are on display in showcases set artistically into the walls of the lounge. The club serves as a place where

these and other sponsors of exhibits in the main exhibition halls may meet members of the medical and allied professions under pleasant circumstances.

The medical and public-health exhibit, being both scientific and educational, comprises probably the largest single enterprise of its kind ever undertaken specifically for adult health education. The exhibit is in two sections. A vast Hall of Man, which sets forth in unique fashion essential information on human anatomy and physiology, is under sponsorship of the American Museum of Health, with generous assistance from a number of philanthropic foundations and public-spirited life insurance companies and commercial institutions. Adjoining the Hall of Man is the Hall of Medical Science and Public Health, an outstanding collection of exhibits on such subjects as tuberculosis, pneumonia, syphilis and maternity and child health.

Local physicians, public-health workers and allied professionals will utilize the club to entertain out-of-town guests brought here by the many meetings of national and international groups to be held in New York during the fair. Members of the International Congress of Microbiology meeting in September, 1939, to use one example, may turn to the club not only for information regarding the medical and public-health resources in and about New York City, but for guidance in seeking authoritative local sources of information on the latest developments in technical aspects of their specialized fields.

Management of the club is vested in a board of directors which includes officers of the county medical societies of the five boroughs constituting the City of New York and of adjacent Westchester and Nassau counties. It is a non-profit membership organization, incorporated under the laws of New York State.

NOTES

Among the fifteen scientists elected to membership at the annual meeting of the National Academy of Scientists were Dr. William B. Castle and Dr. A. Baird Hastings, of Boston. The former is professor of medicine and the latter Hamilton Kuhn Professor of Biological Chemistry at the Harvard Medical School.

Dr. Leroy M. S. Miner, dean of the Harvard Dental School and former president of the American Dental Association, was recently awarded the Jarvie Fellowship Medal by the Dental Society of the State of New York. This medal is presented each year in recognition of outstanding contributions to the dental profession.

CORRESPONDENCE

SELECTION OF ORATOR

To the Editor: I note in the proposed amendments to the by-laws of the Massachusetts Medical Society, a suggested change in Chapter IV, Section 3, which limits the selection of the orator for the annual meeting to members of the Council. It is very difficult for me to see any rationale for such limitation. I assume that the society wishes for its annual orator the most effective and capable man for this particular job. It is perfectly reasonable that the most desirable man would usually be a member of the Council, but this does not necessarily follow. For these reasons I should question the desirability of this part of the amendment.

HENRY A. CHRISTIAN, M.D.

721 Huntington Avenue,
Boston.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

To the Editor: In addition to the articles enumerated in our letter of March 31 the following have been accepted:

Abbott Laboratories

Tablets Barbital Sodium — Abbott, 5 gr.

Armour Laboratories

Suprarenalin Solution 1:1000 in 1 cc. Ampules (for hypodermic use)
Suprarenalin Solution 1:1000 in 10 cc. Vials (for hypodermic use)
Suprarenalin Solution 1:1000 in 1 oz. Bottles (for hypodermic use)

Cutter Laboratories

Ampules Iodobismitol with Saligenin, 2 cc.

Gane's Chemical Works, Inc.

Racephedrine Hydrochloride

The National Drug Co.

Undulant Fever Vaccine (Abortus and Suis)
Undulant Fever Vaccine (Melitensis)

Sharp & Dohme

Antipneumococcic Serum Type II, Refined and Concentrated — Mulford
Antipneumococcic Serum Type VII, Refined and Concentrated — Mulford

The Upjohn Company

Ampule Solution Sodium Morrhuate 5 per cent with Benzyl Alcohol 2 per cent, 2 cc.
Solution Sodium Morrhuate 5 per cent with Benzyl Alcohol 2 per cent, 30 cc. vials
Ampule Solution Sodium Morrhuate 10 per cent with Benzyl Alcohol 2 per cent, 2 cc.
Solution Sodium Morrhuate 10 per cent with Benzyl Alcohol 2 per cent, 30 cc. vials
Tablets Sulfanilamide, 5 gr.
Tablets Sulfanilamide, 7½ gr.

PAUL NICHOLAS LEECH, *Secretary.*

535 North Dearborn Street,
Chicago, Illinois.

REPORTS OF MEETINGS

HOSPITAL RESEARCH COUNCIL

A meeting of the Hospital Research Council of the Massachusetts General Hospital was held on Tuesday, January 31, in the Ether Dome of the Massachusetts General Hospital, Dr. Arthur W. Allen presiding.

The program was opened by Dr. Samuel Hertz, who spoke on "Radioactive Iodine." Dr. Hertz began by describing the method of measurement, using the Geiger counter, to record the amount of radioactivity emanated. The electrons set off a high voltage spark across a gap in a chamber containing air under reduced pressure by ionizing this air. The electrons pass in through a window in the chamber. The electrical discharges are converted into sounds, which can be counted and recorded. The amount of radioactivity in a test substance, then, is in proportion to the number of sounds given off by the counter.

There are five known isotopes of iodine and probably more which have not been identified. These can be made radioactive by subjecting them to bombardment from a

neutron source. The course of such a radioactivated compound after injection can be traced in its distribution to the various tissues in the body. The experiment consisted of injecting radioactive iodine compounds into rabbits, which had been treated previously with anterior pituitary extract to render the thyroid gland hyperplastic. Fifteen minutes later the rabbits were killed and the various tissues tested with the Geiger counter.

It was found that the thyroid gland contained 38 arbitrary units, whereas the spleen contained only 2, the liver 4, the pituitary gland 0, muscle 0, and the urine 48 (due to the excretion of the iodide). Dr. Hertz presented a chart showing the rate of absorption by various types of thyroid gland. The markedly hyperplastic gland is much more able to attract the iodine than is the normal thyroid. After the first fifteen or twenty minutes only a small amount of iodide is further attracted. Dr. Hertz suggested that this might prove to have applications of distinct value in estimating thyroid disorders.

This work was undertaken in collaboration by the Massachusetts Institute of Technology and the Thyroid Clinic of the Massachusetts General Hospital. The discussion further revealed that a good many chemical and biological substances can be made radioactive.

The next paper, on "Prostigmin," was presented by Dr. Henry R. Viets. Dr. Viets reviewed the history of myasthenia gravis. It is a very rare disease, the first case at the Massachusetts General Hospital having been seen in 1905. From 1905 to 1935, 30 clear-cut cases were recorded. A quarter of these died in the hospital, the fate of the remainder being largely unknown. Many probably died within two to five years after being seen. Since 1935 there have been 44 cases treated at the hospital, and 6 or 10 more outside. This sudden jump in admissions is the result of the discovery of the action of the drug, Prostigmin, in 1934; it relieved the principal symptoms almost immediately.

Textbooks to the contrary, myasthenia gravis is not a disease of young people; it can occur in persons aged sixty or seventy. The disease is as serious as it ever was, but the mortality has been markedly reduced following treatment with Prostigmin. Other drugs are by no means as effective. The disease is characterized by remissions and relapses; the drug tides over the relapses to stages of remission, thus saving lives. Physiological studies are to be carried out in the near future.

Dr. Arturo Rosenblueth, in opening the discussion of Dr. Viets's paper, reported some work on the physiology of the disease. An important step in the transmission of stimuli from motor nerve to muscle is the release of acetylcholine at the neuromuscular junction. Wherever acetylcholine is thus liberated, there must be an enzyme, esterase, that splits it to form acetic acid and choline, releasing its effect. Prostigmin prevents or stops the action of this esterase. The question is whether myasthenia gravis is due to an amount of acetylcholine below the required threshold or to a disturbance and abnormal production of esterase. Prostigmin, then, either protects the acetylcholine or inhibits the excess production of esterase.

Dr. Otto Krayer brought up the question of similarity between myasthenia gravis and curare poisoning and overdosage with the magnesium ion. In reply to Dr. Krayer's question about dosage of Prostigmin, Dr. Viets stated that he found the minimum effective intramuscular dose to be 4.5 mg., and 120 mg. by mouth. The difference in the amounts in the two forms of administration is unexplained.

The third paper was presented by Dr. Tracy B. Mallory: "The Development of Emphysema in Chronic Bronchial Asthma." Dr. Mallory said that it was first necessary to define emphysema, since clinicians and pathologists have always differed. He classified emphysema into three types. The first or earliest he called "physiologic emphysema," occurring under exertion, or in asthma during attacks only. The next stage-type was "functional emphysema," which is permanent and physiologic. The third was "structural emphysema," characterized by actual anatomic changes in the lung parenchyma: fusion of air sacs, rupture of alveolar walls and diminution of the respiratory surface area.

Dr. Mallory analyzed 45 cases that had come to autopsy. Many of these patients had had asthma without emphysema. There were 13 cases demonstrating active organizing pneumonia or the resultant scarification. Thus, patches of lung were contracted to scars and the surrounding alveoli were dilated so that actual lung volume was unchanged. The more severe the process the more likely it is for bullae to develop as alveolar walls break. The primary causes of death in the group of 45 cases included 12 of pneumonia, 13 of paroxysmal attack, 4 of right-heart failure and 2 of pulmonary insufficiency. There were 12 cases of asthma with cor pulmonale, but of these only a few had histologic emphysema. Thirteen cases of the 45 had structural emphysema.

Dr. Mallory summarized his observations as follows: the relation of emphysema to asthma is overemphasized, but the relation of cor pulmonale to asthma is underrated; the development of emphysema depends more on the pneumonias than it does on pure asthmatic attacks.

The last paper, "Studies of Plasma Volume," was read by Dr. Edward Hamlin, Jr. Dr. Hamlin limited his discussion to the effect of adrenalin, Nembutal and sympathectomy on plasma volume in the cat. Plasma-volume determinations were based on dilution values of a dye. After the initial determination, adrenalin (1:1000) was given intravenously in a dose three times the amount sufficient for minimal afferent stimulation. In the first experiment 8 out of 10 unanesthetized cats showed a 5 per cent increase in plasma volume. On repetition, all 10 showed an increase. This corroborates the results obtained by other investigators, but is of greater value because an anesthetic effect did not enter into the picture. In an attempt to evaluate the anesthetic factor, an experiment was carried out with Nembutal, 0.5 cc. per kilogram given to each of 6 cats. These showed an immediate increase in plasma volume of 10 per cent; following adrenalin there was a relative decrease — relative in the sense that it never reached the value of the initial reading in a non-anesthetized cat.

Sympathectomy gave variable increases in plasma volume. In sympathectomized cats, Nembutal produced a slight increase, and adrenalin then effected a marked decrease. Explanation of the phenomena is difficult; the effects are probably due to the action of adrenalin on the spleen and that of the barbiturate on vasodilatation, the mechanism of the plasma-volume shifts being referable to the capillaries and venules.

TRUDEAU SOCIETY

A meeting of the Trudeau Society was held Thursday evening, February 2, in the Beth Israel Hospital auditorium, Dr. Leon A. Alley presiding. Dr. Alley introduced the speaker of the evening, Dr. Edgar Mayer, assistant professor of medicine, Cornell University Medical College, whose subject was "Diet in the Treatment of Tuberculosis."

Dr. Mayer limited his remarks to a consideration of the particular dietary treatment practiced in certain clinics in Vienna and Munich. He had been sent to these clinics

with Dr. Miller on a special commission to study the dietary regimen used in 1928 and thereafter.

Based on the observation that an acid, ash-residue diet promotes better and more rapid healing of wounds than does a neutral or alkaline one, Sauerbruch and Hermannsdorfer attempted to apply these principles to the treatment of tuberculosis. Gerson entered on this investigation and modified the method. The essence of the diet is low salt and high vitamin content. Special equilibrated salts of calcium, magnesium and potassium are given instead of sodium chloride. Fluids are restricted, and cod-liver oil given in large amounts. This diet has the drawback of being very tasteless and difficult to institute.

Gerson's method is to restrict protein intake at first and then gradually give more. His essential is a vegetable basis — raw, boiled, steamed, extracted vegetables and an abundance of fruit and vegetable juices. A Hermannsdorfer diet for a 50-kilo man might consist of the following: 90 gm. of protein, 160 gm. of fat and 240 gm. of carbohydrate (40 to 50 calories per kilo of body weight). In addition the patient is given 40 gm. of cod-liver oil, and the fluid intake is restricted to 1500 cc. daily. Salt is restricted to 2 or 3 gm. daily, the normal ingestion being about 15 gm. With the equilibrated salt, one must give hydrochloric acid. Meat and eggwhite are restricted.

In 1926, Sauerbruch first published a report of such treatment in pulmonary tuberculosis, but an analysis of this report reveals no suitable controls. In 1928 the series had been increased to 168 tuberculous patients by Hermannsdorfer and Sauerbruch, who reported glowing results. These results did not seem to be corroborated by other workers. In 1930 Dr. Mayer tried the treatment at Saranac and was not particularly impressed. In 1932 Hermannsdorfer reported the results on 34 patients, 22 treated and 12 controls. Apparently no profound effect of the diet was demonstrable. Hermannsdorfer claimed that he got no great effect in the "exudative type" of pulmonary tuberculosis; in the "productive type," however, he claimed to have lowered the temperature and to have decreased the amount of sputum. In 1935 further reports were also not convincing on account of poor controls. In summary the dietary treatment of pulmonary tuberculosis seems to have had very rare dramatic results but has not been consistently of benefit. Perhaps a fair trial has not yet been given, certainly not in this country.

In tuberculosis of the bones and joints and in genitourinary tuberculosis the results have been more consistent, although again the controls are not sufficient. Gerson included in his dietary regimen certain "thrust" days, meaning days in which nothing but raw fruits and vegetables and their juices were eaten. This essentially is a high potassium intake. Gerson claims to have obtained marked improvement in his cases; a single significant piece of evidence is that whereas ordinarily a tuberculous joint becomes quite reddened, painful and swollen if massaged, after a "thrust period" the joint may be so treated with impunity.

Dr. Mayer described a few of his own cases in which as a last resort he tried the Gerson diet and apparently got remarkable results. However, he again concluded conservatively that the very few cases with dramatic results do not constitute an average that allows recommendation of the diet treatment per se.

Tuberculosis of the skin and mucous membranes presents some interesting features. It had been shown previously by German investigators that after a period of a salt-free diet, the daily ingestion of 20 gm. of sodium chloride produced without exception an excessive outbreak of skin lesions in several types of skin disease, tuberculous and otherwise. This effect proved to be due to the sodium and not the chloride ion. It was hypothesized that the sodium ion makes the cell membrane more permeable, and hence the cell becomes reactant to stimuli which ordinarily are not effective. From 1928 to 1932, a series of 600 cases of tuberculosis of the skin so treated was reported, with beneficial results. This work has been confirmed. Tuberculosis of the mucous membranes was said to heal in six or eight weeks; lupus-vulgaris nodules began to disappear in from six to ten weeks. One clinic reported 33 cases of lupus vulgaris treated with the Hermannsdorfer diet, including equilibrated calcium, magnesium and potassium salts. By microscopic photography of the capillary and subcapillary plexuses it was shown that the capillaries narrowed and the circulation improved. Identical changes occurred in cases of x-ray dermatitis so treated, unless the lesion had gone on beyond telangiectasia to malignancy.

Dr. Mayer concluded that there is a relation between water economy and reduced intake of salt. Tissue imbibition promotes an inflammatory reaction; therefore dehydration has the opposite effect. Vitamin-rich foods are a defense against the progress of infection. The effect of high intake of the cations "antagonistic" to the sodium ion is as yet still in doubt. Clinically, the effect of the Hermannsdorfer and Gerson diets is also still in doubt. Although a few patients with pulmonary tuberculosis have shown favorable results, the beneficial effect is common in tuberculosis of the bones, joints and genitourinary system and commonest in tuberculosis of the skin. The treatment is expensive and very difficult to administer. He added that there is no specific dietary treatment for tuberculosis but that certain diets will nonspecifically aid by producing beneficial vascular, chemical and cell-permeability changes.

Dr. Leon A. Alley opened the discussion of Dr. Mayer's paper. He described an experiment carried out at Lakeville in conjunction with the Forsyth Dental Clinic which proved fairly conclusively that, in patients with extrapulmonary tuberculosis, a high-vitamin-C diet decreased the incidence of carious teeth and also promoted an improvement in affected bones and joints.

Dr. Mayer answered several questions as to details of his talk by members of the audience. The one significant point brought out was that he offered the Hermannsdorfer or Gerson dietary treatment to his patients only when other accepted measures had failed or could not be carried out. He made a plea for an open mind on this subject.

GREATER BOSTON MEDICAL SOCIETY

A meeting of the Greater Boston Medical Society was held in the auditorium of the Beth Israel Hospital on March 7, with Dr. Louis M. Freedman presiding. Dr. Maxwell Finland, associate in medicine, Harvard Medical School, and assistant physician, Thorndike Memorial Laboratory, Boston City Hospital, was the speaker of the evening, and his subject was "The Present Status of the Specific Treatment of Pneumococcal Pneumonia and Other Pneumococcal Infections."

The speaker retraced the rapid strides in treatment brought about in the past ten years, and particularly in the past two or three years. As long ago as 1910, typing of the pneumococcus was carried out; and three fundamentals of therapy in this disease, which still obtain, were postulated at that time: namely, that the antiserum must be type specific, must give a high titer of antibody and must be given early in the course of the disease.

The important advances in the specific therapy of pneumococcal infections since that time have been the stand-

ardization of antiserums by exact methods; the concentration of serums so that a similar number of antibodies are available in a small volume; the subdivision of Group IV pneumonias and the manufacture of specific serums for some of these higher types; the introduction of therapeutic rabbit serum, in which a higher titer can be obtained in a shorter time than it can in horse serum; and finally chemotherapy, which, while still in its incipiency, appears at least promising.

Dr. Finland presented the results obtained at the Boston City Hospital by all methods of treatment in the pneumonia cases admitted during the ten-year period 1929–1938. It was emphasized that these statistics were complicated not only by the ever-prevalent difficulty in controlling biological experiments, but also by the inadequate amounts of serum available in the earlier years when only the very sick patients were given specific therapy. In regard to etiology, it was pointed out that 80 per cent of all cases of lobar pneumonia in adults are due to Types 1, 2, 3, 5, 7 and 8, with the first three accounting for almost 60 per cent of the whole. On the other hand, although Types 1, 2 and 5 produce lobar pneumonia predominately, the others more often produce atypical pneumonias and focal infections, particularly Type 3.

There are certain generalities which hold for the treatment of pneumococcal infections of whatever type. Thus, bacteremia increases the mortality rate with or without treatment, although the small number of positive blood cultures in the specifically treated patients probably indicates an abortion or prevention of some such cases. The differences in bacteremic and in non-bacteremic mortality rates, however, vary somewhat among the types. A further factor common to all is the efficacy of early treatment, particularly before the fourth day. Moreover, in all types of pneumonia for which there is a specific serum, there has been some reduction in the mortality under treatment. In all these respects there is a quantitative difference in the various types.

The reduction in mortality by the use of specific horse serum varies from 75 per cent in Type 5 to practically nil in Type 3, although all the common types except the latter show at least a 50 per cent drop of the death rate. The apparent failure of Type 3 serum is explained by the paucity of available material and the necessity of saving it for the very sick patients. Rabbit serum finds its greatest use in Types 2 and 7, where the level of antibody titer often reaches five or six times that of horse serum. Although there seems to be no striking reduction in the incidence of empyema following the use of specific therapy, this may be explained by the fact that those who now live to develop this condition would have died without specific treatment, for more patients with pneumonia now avoid a fatal issue.

Untoward reactions from serum therapy occurred in higher percentage in this series of cases than in comparable groups using commercial products, for many of these incidents took place with serums that were being clinically tested before being released for general consumption. This line of reasoning also holds for the high incidence of reactions following the early use of rabbit serum, which was at least as high as that with horse serums. Indeed, the only severe reactions — that is, collapse — occurred in early experimental work. The common manifestations include nausea, vomiting, urticaria, collapse, chills and joint pains. The usual precautions should be taken: a careful allergic history, slow administration of the serum, and constant vigilance during the initial stages of the injection. Administration should be immediately discontinued on noticing any untoward signs, and adrenalin should be used in severe reactions, if necessary.

The common causes of failure in specific serum therapy are: delayed treatment, occasionally due to a misjudgment of the duration of the disease; errors in typing; inadequate or "spread" dosage; focal purulent infections; mixed infections, especially with a streptococcus; and other complicating conditions or diseases, such as pregnancy, nephritis, congestive failure, and so forth.

Dr. Finland went on to discuss the intriguing subject of chemotherapy, but without benefit of conclusive statistics as presented for serum therapy. This discussion was based on the use of sulfanilamide during 1937–1938 in a large series at the Boston City Hospital and observations during the present winter on sulfapyridine. The bacteriostatic action of these drugs in vitro has been proved satisfactory with many pneumococci but is not so striking as it is against the streptococci. And although the range of effectiveness is still debatable, the speaker said that sulfanilamide has exhibited its lifesaving qualities on several occasions. The most promising results have been obtained in Type 3 pneumonias, where sulfapyridine particularly has been extolled. Many preliminary reports on its use compare favorably with results from serum treatment, but none of these have emanated from a clinic where an adequate base line of results with and without serum has been established. Dr. Finland recently reported a group of Type 3 patients treated with serum or sulfanilamide or both and found the results almost identical and seemingly not very striking. However, only the seriously sick patients received sulfanilamide, and since their mortality was at least as low as that of those otherwise treated, some benefit must have resulted from the chemotherapy. These results, together with other clinical reports and in vitro experiments, seem to indicate that in certain instances the optimum results are obtained from a combination of chemotherapy and specific serum therapy. One definite advantage of the addition of these bacteriostatic substances to the regimen is the opportunity it affords of discontinuing serum therapy, with its concomitant dangers, somewhat earlier than heretofore. Untoward reactions are not unheard of with these chemicals, however. Those encountered so far include hemolytic anemia, granulocytopenia, nausea and vomiting, nitrogen retention and certain symptoms of the central nervous system, such as profound depression and wild delirium.

In conclusion, Dr. Finland stated that specific serum therapy has established its worth, with the possibility of rabbit serum, when available, replacing horse serum, due to its greater efficacy per unit volume and greater ease in preparation. The use of sulfanilamide and sulfapyridine, either alone or in combination with serum, appears to offer a powerful means of therapy, but there have not as yet been a sufficient number of controlled studies to substantiate all the roseate claims.

In discussion, Dr. Frederick T. Lord emphasized certain features of the treatment of pneumonia in private practice, especially in regard to serum therapy. Blood culture when the patient is first seen may afford an early and sometimes the only means of typing, besides partly determining the dosage of serum to be given. He finds that, on the whole, too small dosage is given, the administration is too slow, and there is a failure to adjust the dosage to the requirements of the individual case. The nominal amount should be doubled when there is a positive blood culture, when the temperature fails to drop and when there is clinical evidence of a spreading lesion.

Dr. Edward Curnen, of the Children's Hospital, discussed certain differences in the pneumonias of children.

Before the importance of the higher types in the etiology of atypical pneumonias was determined, the specific treatment of pneumococcal infections in children seemed futile, in the face of the remarkably low mortality of lobar pneumonia due to the types common to adults. But, although the replacement of an anatomical classification by an etiologic one has clarified the important place of the higher types of pneumonia in children, Dr. Curnen emphasized the fact that specific treatment is no substitute for good basic supportive handling of young patients. Nevertheless, serum has been tried with gratifying results for the past two years at the Children's Hospital, and the evidence indicates that the course is shortened and the complications decreased. The ease of administration and the fact that mixed infections are favorably treated make chemotherapy a popular form of therapy in serious cases, but statistics are still inconclusive, although they seem to indicate beneficial results.

Dr. William Dameshek reported "amazing" results in 50 per cent of cases of pneumonia treated with sulfapyridine at the Beth Israel Hospital. Indications for its use are a non-typable pneumonia, a type in which specific serum is not available or of equivocal value, a very ill patient, and atypical cases where the typing is questionable. Contraindications include nephritis and a falling white-blood-cell count.

NOTICES

REMOVALS

HAROLD BOWDITCH, M.D., announces the removal of his office to 44 Harvard Avenue, Brookline. Telephone LONgwood 4995.

WILLIAM M. SANTORO, M.D., announces the removal of his office to 1586 Beacon Street, Brookline. Telephone LONgwood 2060.

GREATER BOSTON MEDICAL SOCIETY

The annual dinner meeting of the Greater Boston Medical Society will be held at the University Club, Boston, on Monday, May 22, at 6:30 p. m. Judge Abraham E. Pinanski, of the Superior Court of Massachusetts, will be the speaker.

LOUIS M. FREEDMAN, M.D., *President,*
DAVID B. STEARNS, M.D., *Secretary.*

TRUDEAU SOCIETY

The annual meeting of the Trudeau Society will be held at the Plymouth County Sanatorium, on Thursday, May 25, at 4:00 p. m. Dr. Benjamin Gruskin of Temple University, Philadelphia, will speak on "The Intracutaneous Method for the Determination of Activity in Cases of Pulmonary Tuberculosis." Dr. Lauren V. Ackerman, of Rutland State Sanatorium, will report on a case of chronic cor pulmonale with unusual pathological findings.

There will be election of officers.

MOSES J. STONE, M.D., *Secretary.*

NEW ENGLAND SOCIETY OF PHYSICAL MEDICINE

The annual meeting of the New England Society of Physical Medicine will be held on Wednesday evening, May 24, at the Hotel Kenmore, Boston.

The Council will meet at 6:00, and an informal dinner will be held in the Empire Room at 6:30. The program will begin at 7:45.

PROGRAM

Investigation of Certain Problems in Relation to Tissue Heating. Dr. Michael Pijoan.

Dangers and Complications. Dr. Herman A. Osgood.

Short-Wave Therapy in Arthritis and Bursitis. Dr. Heinrich G. Brugsch.

Clinical Evaluation of Short-Wave Therapy. Dr. William D. McFee.

All members of the medical profession are cordially invited to attend.

WILLIAM D. MCFEE, M.D., *Secretary.*

FOURTH ANNUAL CONVENTION OF THE NATIONAL GASTROENTEROLOGICAL ASSOCIATION

The fourth annual convention of the National Gastroenterological Association will be held at Squibb Hall, Squibb Building, 745 Fifth Avenue, New York City, on June 1 and 2. A very interesting program is assured.

Members of the medical profession are cordially invited to attend.

HENRY KENDALL, M.D., *Chairman,*
Program Committee.

AMERICAN CONGRESS OF PHYSICAL THERAPY

The eighteenth annual scientific and clinical session of the American Congress of Physical Therapy will be held on September 5, 6, 7 and 8 at the Hotel Pennsylvania, New York City. Preceding these sessions the Congress will conduct an intensive instruction seminar in physical therapy for physicians and technicians — August 30 and 31 and September 1 and 2.

For information concerning the seminar and the preliminary program of the convention proper, address the American Congress of Physical Therapy, 30 North Michigan Avenue, Chicago.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, MAY 22

MONDAY, MAY 22

6:30 p. m. Greater Boston Medical Society. University Club, Boston.

TUESDAY, MAY 23

*9-10 a. m. Hemoglobin, Iron, Bilirubin. Dr. George Barkan. Joseph H. Pratt Diagnostic Hospital.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

WEDNESDAY, MAY 24

*9-10 a. m. Hospital case presentation. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*12 m. Clinicopathological conference. Children's Hospital amphitheater.

*7:45 p. m. New England Society of Physical Medicine. Hotel Kenmore, Boston.

THURSDAY, MAY 25

*9-10 a. m. Rheumatism: Presentation of cases. Dr. Walter Bauer. Joseph H. Pratt Diagnostic Hospital.

FRIDAY, MAY 26

*9-10 a. m. Recent Advances in the Understanding of Gastric Secretion: Experimental and clinical observations. Dr. Oliver Cope. Joseph H. Pratt Diagnostic Hospital.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

12 m. Clinical meeting of the Children's Medical Service. Massachusetts General Hospital. Ether Dome.

*9 p. m. Massachusetts Italian Medical Society. Hotel Kenmore, Boston.

SATURDAY, MAY 27

*9-10 a. m. Hospital case presentation. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*10 a. m. - 12 m. Staff rounds of the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.

MAY 19 — Boston Dispensary. Luncheon meeting of the Clinical Staff. Page 817, issue of May 11.

MAY 22 — Greater Boston Medical Society. Page 857.

MAY 22, 23, and 24 — American Association for the Study of Goiter. Page 405, issue of March 2.

MAY 24 — New England Obstetrical and Gynecological Society. Page 817, issue of May 11.

MAY 24 — New England Society of Physical Medicine. Page 857.

MAY 25 — Trudeau Society. Page 857.

MAY 26 — Massachusetts Psychiatric Society. Page 817, issue of May 11.

MAY 26 — Massachusetts Italian Medical Society. Page 768, issue of May 4.

JUNE 1 and 2 — Fourth Annual Convention of the National Gastroenterological Association. Page 857.

JUNE 5, 6, 7, and 8 — American Association of Industrial Physicians and Surgeons. Page 581, issue of March 30.

JUNE 6 — Harvard Medical Alumni Association. Page 851.

JUNE 6 — Tufts College Medical School Alumni. Page 851.

JUNE 6 — Boston University School of Medicine Alumni Association. Page 851.

JUNE 6, 7, and 8 — Massachusetts Medical Society. Worcester.

JUNE 7 — Massachusetts Medico-Legal Society. Page 851.

JUNE 12-17 — Symposium on the Public-Health Significance of the Virus and Rickettsial Diseases. Page 815, issue of May 11.

JUNE 26-29 — National Tuberculosis Association. Page 936, issue of December 8.

JUNE 29 — Pentucket Association of Physicians, 8:30 p. m., Hotel Bartlett, 95 Main Street, Haverhill.

AUGUST 30 - SEPTEMBER 2 — Seminar in Physical Therapy. Page 857.

SEPTEMBER — Boston Psychoanalytic Institute. Page 450, issue of September 22.

SEPTEMBER 5-8 — American Congress of Physical Therapy. Page 857.

SEPTEMBER 11-15 — American Congress on Obstetrics and Gynecology. Page 938, issue of December 8.

SEPTEMBER 15-28 — Pan-Pacific Surgical Association. Page 863, issue of November 24.

OCTOBER 23 - NOVEMBER 3 — New York Academy of Medicine. Page 581, issue of March 30.

FALL, 1939 — Temperature Symposium. Page 218, issue of February 2.

BOOK REVIEWS

A Bibliography of the Writings of Harvey Cushing: Prepared on the occasion of his seventieth birthday, April 8, 1939. The Harvey Cushing Society. 108 pp. Springfield, Illinois: Charles C Thomas, 1939. $5.00.

This beautifully printed book, complete in every sense, is a welcome addition to medical literature and a fitting *Festschrift* for one of America's greatest surgeons. It contains the facts of his life, his degrees, a list of each monograph and paper by him, as well as those coming from his clinic, and a list of his associates. There are six hundred and fifty-eight items in all, cited with care and augmented with additional notes, giving unusual facts about the articles. The book is worthy of the man. There is an appropriate introduction by Arnold C. Klebs. A limited edition, with special typographical features, has been published by the Harvey Cushing Society. The compiler and the printer have worked together to produce a book of exceptional value.

The Extra Pharmacopoeia. Martindale. Twenty-first edition. Vol. 2. 1148 pp. London: The Pharmaceutical Press, 1938. 22s. 6d.

When the production of a book extends through twenty-one editions it is fair to assume that there is approval of its quality to interest and instruct its readers. In this publication, the Council of the Pharmaceutical Society of Great Britain presents the advances made in pharmacology and the ancillary subjects of medical knowledge.

Beginning with an analytical addendum to the list of chemicals and the materia medica set forth in Volume 1 of this edition, there will be found in subsequent sections of this volume a list of the proprietary medicines sold in Great Britain with the composition of each, so far as ascertained; following are chapters on reagents and analyses of body fluids, nutrition and vitamins, chemical substances used in therapeutics, and bacteriological and clinical notes in explanation of an extensive list of diseases, with the preventive and therapeutic principles applicable to each.

The wide range of subjects dealt with requires an extensive, carefully and concisely phrased text which conveys useful up-to-date information, for physicians and students, relating to the preservation of health and the alleviation of human suffering.

Dunant: The story of the Red Cross. Martin Gumpert. 323 pp. New York: Oxford University Press, 1938. $2.50.

This is a well-written book concerning a strange and interesting man. It is more than a biography for the author not only gives the facts of Dunant's life but also records the history of Europe from 1850 to 1910, the year of Dunant's death. After the battle of Solferino in 1859, where Dunant first visualized the need for a Red Cross in war, one travels with this extraordinary man from country to country, watching the growth of the Red Cross idea on the background of European political, martial and economic upheavals. The author has done his work well, and the book gives a splendid picture of the man and his times. Dunant's career as one of the most distinguished men of his day, the Nobel prize of 1901, his loss of friends and final poverty, and his tragic end in 1910, are all clearly told. The text appears to be well translated from the German.

Allergic Diseases: Their diagnosis and treatment. Ray M. Balyeat and Ralph Bowen. Fifth edition. 547 pp. Philadelphia: F. A. Davis Co., 1938. $6.00.

A clear conception of allergic manifestations has been a much needed chapter in medical knowledge of today. This need has been met by a perfectly clear, lucid and comprehensive text written by Dr. Balyeat. The matter is comprehensively and systematically organized and presents the allergic manifestations of almost every known substance. It also explains the errors in the reactions and proposes the need of a conservative judgment on the part of the physician. The plates reproduced in the text, however, are particularly poor, being dark and with little detail. In spite of this, the book must be highly recommended to every internist who needs a handbook and text for reference to the problem of allergy.